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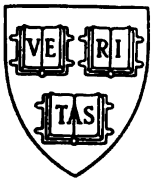

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OCTOBER, 1886.

Those marked thus (†) have paid the Composition Fee in lieu of further annual subscriptions.

Amongst the non-residents those marked thus (*) are entitled by composition to receive the Transactions.

Elected

1846 *ABERCROMBIE, JOHN, M.D.

1877 ABERCROMBIE, JOHN, M.D., Assistant Physician to, and Lecturer on Forensic Medicine at, Charing Cross Hospital; 23, Upper Wimpole street, Cavendish square.

1885 ABRAHAM, PHINEAS S., 40, Elgin Road, St. Peter's Park.

1851 *ACLAND, SIR HENRY WENTWORTH, K.C.B., M.D., LL.D., F.R.S., Honorary Physician to H.R.H. the Prince of Wales; Regius Professor of Medicine in the University of Oxford; Broad street, Oxford.

Elected

1885 ACLAND, THEODORE DYKE, M.D., Assistant Physician to the Hospital for Consumption and Diseases of the Chest, Brompton; 7, Brook street, Hanover square.

1847 ACOSTA, ELISHA, M.D., 24, Rue de Luxembourg, St. Honoré, Paris.

1852 †ADAMS, WILLIAM, Surgeon to the Great Northern Hospital and to the National Hospital for the Paralysed and Epileptic; Consulting Surgeon to the National Orthopædic Hospital, Great Portland street; 5, Henrietta street, Cavendish square. C. 1873-4. *Trans.* 3.

1867 AIKIN, CHARLES ARTHUR, 7, Clifton place, Hyde park.

1837 *AINSWORTH, RALPH FAWSETT, M.D., Consulting Physician to the Manchester Royal Infirmary; Cliff Point, Lower Broughton, Manchester.

1839 ALCOCK, SIR RUTHERFORD, K.C.B., K.C.T., K.T.S., D.C.L., late H.M.'s Envoy Extraordinary at the Court of Pekin. *Trans.* 1.

1866 ALLBUTT, THOMAS CLIFFORD, A.M., M.D., F.R.S., Physician to the Leeds General Infirmary; 35, Park square, Leeds. *Trans.* 3.

1879 ALLCHIN, WILLIAM HENRY, M.B., F.R.S. Ed., Physician to, and Lecturer on Medicine at, the Westminster Hospital; 5, Chandos street, Cavendish square, W.

1863 ALTHAUS, JULIUS, M.D., Senior Physician to the Hospital for Epilepsy and Paralysis, Regent's park; 48, Harley street, Cavendish square. *Trans.* 2.

1884 ANDERSON, ALEXANDER RICHARD, Resident Surgeon, General Hospital, Nottingham.

1881 ANDERSON, JAMES, A.M., M.D., 84, Wimpole street, Cavendish square.

1862 ANDREW, EDWYN, M.D., 12, St. John's Hill, Shrewsbury.

1862 ANDREW, JAMES, M.D., Physician to, and Lecturer on Medicine at, St. Bartholomew's Hospital; 22, Harley street, Cavendish square. S. 1878-9. C. 1881-2. *Trans.* 1.

1820 ANDREWS, THOMAS, M.D., Norfolk, Virginia.

1880 *APPLETON, HENRY, M.D., Staines.

Elected

1874 AVELING, JAMES H., M.D., Physician to the Chelsea Hospital for Women; 1, Upper Wimpole street, Cavendish square.

1851 *BAKER, ALFRED, Consulting Surgeon to the Birmingham General Hospital; 3, Waterloo street, Birmingham.

1873 *BAKER, J. WRIGHT, Senior Surgeon to the Derbyshire General Infirmary; 102, Friargate, Derby.

1865 BAKER, WILLIAM MORRANT, Surgeon to, and Lecturer on Physiology at, St. Bartholomew's Hospital; Consulting Surgeon to the Evelina Hospital for Sick Children; Examiner in Surgery at the University of London; 26, Wimpole street, Cavendish square. C. 1878-9. *Referee*, 1886. *Lib. Com.* 1876-7. *Trans.* 7.

1869 BAKEWELL, ROBERT HALL, M.D., Ross, Westland, New Zealand.

1839 †BALFOUR, THOMAS GRAHAM, M.D., F.R.S., Surgeon General; Coombe Lodge, Wimbledon Park. C. 1852-3. V.P. 1860-1. T. 1872. *Lib. Com.* 1849. *Trans.* 2.

1885 BALLANCE, CHARLES ALFRED, M.S., 56, Harley street, Cavendish square. *Trans.* 1.

1848 †BALLARD, EDWARD, M.D., Inspector, Medical Department, Local Government Board; 12, Highbury terrace, Islington. C. 1872. V.P. 1875-6. *Referee*, 1853-71. *Lib. Com.* 1855. *Trans.* 5.

1866 *BANKS, JOHN THOMAS, M.D., Physician in Ordinary to the Queen in Ireland; Physician to Richmond, Whitworth, and Hardwicke Hospitals; Regius Professor of Physic in the University of Dublin; Member of the Senate of the Queen's University in Ireland; 45, Merrion square, Dublin.

1879 BARKER, ARTHUR EDWARD JAMES, Surgeon to University College Hospital, and Assistant Professor of Clinical Surgery and Teacher of Practical Surgery at University College, London; 87, Harley street, Cavendish square *Trans.* 4.

Elected

1882 BARKER, FREDERICK CHARLES, M.D., Surgeon-Major, Bombay Medical Service [care of ARTHUR E. J. BARKER, 87, Harley street].

1833 †BARKER, THOMAS ALFRED, M.D., Consulting Physician to St. Thomas's Hospital; 109, Gloucester place, Portman square. C. 1844-5. V.P. 1853-4. T. 1860-2. *Referee*, 1848-51. *Trans.* 6.

1876 BARLOW, THOMAS, M.D., B.S., Physician to University College Hospital; Physician to the Hospital for Sick Children, Great Ormond street, and Assistant Physician to the London Fever Hospital; 10, Montague street, Russell square. *Trans.* 1.

1881 *BARNES, HENRY, M.D., F.R S. Ed., Physician to the Cumberland Infirmary; 6, Portland square, Carlisle.

1861 BARNES, ROBERT, M.D., 15, Harley street, Cavendish square. C. 1877-8. *Referee*, 1867-76. *Lib. Com.* 1869-73. *Trans.* 4.

1864 BARRATT, JOSEPH GILLMAN, M.D., 8, Cleveland gardens, Bayswater.

1880 BARROW, A. BOYCE, Assistant Surgeon to King's College Hospital, to the Westminster Hospital, and to the West London Hospital; 17, Welbeck street, Cavendish square.

1840 BARROW, BENJAMIN, Surgeon to the Royal Isle of Wight Infirmary; Southlands, Ryde, Isle of Wight.

1859 BARWELL, RICHARD, Surgeon to, and Lecturer on Surgery at, the Charing Cross Hospital; 55, Wimpole street, C. 1876-77. V.P. 1883-4. *Referee*, 1868-75, 1879-82. *Trans.* 11.

1868 BASTIAN, HENRY CHARLTON, M.A., M.D., F.R.S., Professor of Clinical Medicine and of Pathological Anatomy in University College, London; Physician to University College Hospital and to the National Hospital for the Paralysed and Epileptic; 20, Queen Anne street, Cavendish square. *Referee*, 1886. C. 1885. *Trans.* 1.

1875 BEACH, FLETCHER, M.B., Medical Superintendent, Metropolitan District Asylum, Darenth, near Dartford, Kent.

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1883 BEALE, EDWIN CLIFFORD, M.A., M.B., Assistant Physician to the City of London Hospital for Diseases of the Chest; and Physician to the Great Northern Hospital; 23, Upper Berkeley street.

1862 BEALE, LIONEL SMITH, M.B., F.R.S., Professor of the Principles and Practice of Medicine in King's College, London, and Physician to King's College Hospital; 61, Grosvenor street. C. 1876-77. *Referee*, 1873-5. *Trans.* 1.

1860 *BEALEY, ADAM, M.D., M.A., Oak Lea, Harrogate.

1856 BEARDSLEY, AMOS, F.L.S., Bay villa, Grange-over-Sands, Lancashire.

1871 BECK, MARCUS, M.S., Professor of Surgery in University College, London, and Surgeon to University College Hospital; 30, Wimpole street, Cavendish square. C. 1886. *Referee*, 1882-5. *Lib. Com.* 1881-5.

1880 BEEVOR, CHARLES EDWARD, M.D., Assistant Physician to the National Hospital for the Paralysed and Epileptic; 33, Harley street, Cavendish square. *Trans.* 1.

1858 BEGLEY, WILLIAM CHAPMAN, A.M., M.D., late of the Middlesex County Lunatic Asylum, Hanwell; 26, Saint Peter's square, Hammersmith. C. 1877–8.

1871 BELLAMY, EDWARD, Surgeon to, and Lecturer on Anatomy at, Charing Cross Hospital; Lecturer on Artistic Anatomy to the Science and Art Department, South Kensington; Examiner in Surgery in the Victoria University, Manchester; 17, Wimpole street, Cavendish square. C. 1886. *Referee*, 1882-5. *Lib. Com.* 1879-81. *Trans.* 1.

1847 BENNET, JAMES HENRY, M.D., The Ferns, Weybridge, and Mentone.

1880 BENNETT, ALEX. HUGHES, M.D., Assistant Physician to the Westminster Hospital; 76, Wimpole street, Cavendish square. *Trans.* 1.

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1883 BENNETT, STORER, Dental Surgeon to, and Lecturer on Dental Surgery at, the Middlesex Hospital; Dental Surgeon to the Dental Hospital of London; 17, George street, Hanover square.

1877 BENNETT, WILLIAM HENRY, Assistant Surgeon to, and Lecturer on Anatomy at, St. George's Hospital; 1, Chesterfield street, Mayfair.

1845 †BERRY, EDWARD UNWIN, 17, Sherriff road, West Hampstead.

1885 BERRY, JAMES, Assistant Demonstrator of Anatomy, St. Bartholomew's Hospital; 27, Upper Bedford place.

1820 BERTIN, STEPHEN, Paris.

1872 BEVERLEY, MICHAEL, M.D., Assistant Surgeon to the Norfolk and Norwich Hospital; 63, St. Giles's street, Norwich.

1865 *BICKERSTETH, EDWARD ROBERT, Surgeon to the Liverpool Royal Infirmary, and Lecturer on Clinical Surgery in the Liverpool Royal Infirmary School of Medicine; 2, Rodney street, Liverpool. *Trans.* 1.

1878 BINDON, WILLIAM JOHN VEREKER, M.D., 48, St. Ann's street, Manchester.

1854 BIRD, PETER HINCKES, F.L.S.

1856 BIRD, WILLIAM, Consulting Surgeon to the West London Hospital; Bute House, Hammersmith.

1849 †BIRKETT, EDMUND LLOYD, M.D., Consulting Physician to the City of London Hospital for Diseases of the Chest; 48, Russell square. C. 1865-6. *Referee*, 1851-9.

1851 †BIRKETT, JOHN, F.L.S., Consulting Surgeon to Guy's Hospital; Corresponding Member of the "Société de Chirurgie" of Paris; Inspector of Anatomy for the Provinces in England and Wales; 62, Green street, Grosvenor square. L. 1856-7. S. 1863-5. C. 1867-8. T. 1870-78. V.P. 1879-80. *Referee*, 1851-5, 1866, 1869, *Sci. Com.* 1863. *Lib. Com.* 1852. *Trans.* 8.

1866 BISHOP, EDWARD, M.D.

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1881 Biss, Cecil Yates, M.D., Assistant Physician to the Hospital for Consumption, Brompton, and to the Middlesex Hospital; 135, Harley street, Cavendish square. *Trans.* 1.

1865 Blanchet, Hilarion, Examiner to the College of Physicians and Surgeons, Lower Canada; 6, Palace street, Quebec, Canada east.

1865 Blandford, George Fielding, M.D., Lecturer on Psychological Medicine at St. George's Hospital; 71, Grosvenor street. C. 1883-4.

1867 Bloxam, John Astley, Surgeon to, and Teacher of Operative Surgery in, Charing Cross Hospital; Surgeon for Out-Patients to the Lock Hospital; Junior Surgeon to the West London Hospital; 8, George street, Hanover square.

1823 Bojanus, Louis Henry, M.D., Wilna.

1846 †Bostock, John Ashton, C.B., Hon. Surgeon to H.M. the Queen; Surgeon-Major, Scots Fusilier Guards; 73, Onslow gardens, Brompton. C.1861-2. V.P. 1870-71. *Sci. Com.* 1867.

1869 Bourne, Walter, M.D. [care of the National Bank of India, 80, King William street, City]; Archaco, France.

1882 Bowlby, Anthony Alfred, Surgical Registrar to St. Bartholomew's Hospital; 75, Warrington crescent, Maida hill. *Trans.* 1.

1870 *Bowles, Robert Leamon, M.D., 8, West terrace, Folkestone.

1841 †Bowman, Sir William, Bart., LL.D., F.R.S., F.L.S., Consulting Surgeon to the Royal London Ophthalmic Hospital, Moorfields; 5, Clifford street, Bond street. C. 1852-3. V.P. 1862. *Referee*, 1845-50, 1854-6. *Lib. Com.* 1847. *Trans.* 3.

1884 Boyd, Stanley, M.B., Assistant Surgeon to the Charing Cross Hospital; 27, Gower street.

1862 Brace, William Henry, M.D., 7, Queen's Gate terrace, Kensington.

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1874 BRADSHAW, A. F., Surgeon-Major; Surgeon to the Rt. Hon. the Commander in Chief in India; Army Head Quarters, Bengal Presidency. [Agent: Vesey W. Holt, 17, Whitehall place.]

1883 BRADSHAW, JAMES DIXON, M.B., 30, George Street, Hanover square.

1867 *BRETT, ALFRED T., M.D., Watford, Herts.

1876 BRIDGES, ROBERT, M.B., Manor House, Yattendon, Newbury, Berks.

1867 BRIDGEWATER, THOMAS, M.B., Harrow-on-the-Hill, Middlesex.

1868 BROADBENT, WILLIAM HENRY, M.D., Physician to, and Lecturer on Medicine at, St. Mary's Hospital; Consulting Physician to the London Fever Hospital; Examiner in Medicine at the University of London; 34, Seymour street, Portman square. C. 1885. *Referee*, 1881-4. *Trans.* 5.

1851 †BRODHURST, BERNARD EDWARD, F.L.S., Surgeon to the Royal Orthopædic Hospital; 20, Grosvenor street. C. 1868-9. *Lib. Com.* 1862-3. *Trans.* 2. *Pro.* 1.

1872 BRODIE, GEORGE BERNARD, M.D., Consulting Physician-Accoucheur to Queen Charlotte's Hospital; 8, Chesterfield street, Mayfair. *Trans.* 1.

1860 BROWN-SÉQUARD, CHARLES EDOUARD, M.D., LL.D., F.R.S., Laureate of the Academy of Sciences of Paris; Professor of Medicine at the College of France; Professor of General Physiology at the Museum of Natural History; Paris. *Sci. Com.* 1862.

1878 BROWNE, Sir JAMES CRICHTON, M.D., LL.D., F.R.S., Lord Chancellor's Visitor in Lunacy; 7, Cumberland Terrace, Regent's Park.

1880 BROWNE, JAMES WILLIAM, M.B., 8, Norland place, Holland Park.

1881 BROWNE, JOHN WALTON, M.D., Surgeon to the Belfast Ophthalmological Hospital; 10, College square N., Belfast.

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1881 BROWNE, OSWALD A., M.A., M.B., Casualty Physician to St. Bartholomew's Hospital and Physician to the Royal Hospital for Diseases of the Chest; 30A, George street, Hanover square.

1874 BRUCE, JOHN MITCHELL, M.D., Physician to, and Lecturer on Materia Medica at, the Charing Cross Hospital; Assistant Physician to the Hospital for Consumption, Brompton; 70, Harley street. *Referee*, 1886. *Trans.* 1.

1871 BRUNTON, THOMAS LAUDER, M.D., F.R.S., Assistant Physician to, and Lecturer on Materia Medica and Therapeutics at, St. Bartholomew's Hospital; Examiner in Materia Medica in the University of London; 50, Welbeck street, Cavendish square. *Referee*, 1880-86. *Lib. Com.* 1882-6.

1860 BRYANT, THOMAS, *Vice-President*, Surgeon to, and Lecturer on Surgery at, Guy's Hospital; 53, Upper Brook street, Grosvenor square. C. 1873-4. V. P. 1885-6. *Sci. Com.* 1863. *Referee*, 1882-4. *Lib. Com.* 1868-71. *Trans.* 10. *Pro.* 1.

1855 BRYANT, WALTER JOHN, Consulting Physician to the Home for Incurable Children, Maida vale; 23A, Sussex square, Hyde park gardens.

1823 BUCHANAN, B. BARTLET, M.D.

1864 BUCHANAN, GEORGE, M.D., F.R.S., Medical Officer of the Local Government Board; Member of the Senate of the University of London; 24, Nottingahm place, Marylebone road.

1864 BUCKLE, FLEETWOOD, M.D.

1876 BUCKNILL, JOHN CHARLES, M.D., F.R.S.; E 2, The Albany, Piccadilly, and Hill Morton Hall, Rugby.

1881 BULLER, AUDLEY CECIL, M.D., Oxford and Cambridge Club, Pall Mall.

1833 †BURROWS, SIR GEORGE, Bart., M.D., D.C.L., LL.D., F.R.S., Physician in Ordinary to H.M. the Queen; Consulting Physician to St. Bartholomew's Hospital; Member of the Senate of the University of London; 18, Cavendish square. C. 1839-40, 1858-9. T. 1845-7. V. P. 1849-50. P. 1869-70. *Referee*, 1842-6, 1850-7, 1861-68, 1875-81. *Lib. Com.* 1836. *Trans.* 2.

Elected

1885 BUTLER-SMYTHE, ALBERT CHARLES, Senior Surgeon to the Grosvenor Hospital for Women and Children; 35, Brook street, Grosvenor square.

1873 BUTLIN, HENRY TRENTHAM, Assistant Surgeon to, and Demonstrator of Practical Surgery and of Diseases of the Larynx at, St. Bartholomew's Hospital; 47, Queen Anne street, Cavendish square. *Trans.* 3.

1871 BUTT, WILLIAM F., 48, Park street, Park lane.

1883 BUXTON, DUDLEY WILMOT, M.D., B.S., 82, Mortimer street, Cavendish square.

1868 BUZZARD, THOMAS, M.D., Physician to the National Hospital for the Paralysed and Epileptic; 56, Grosvenor street, Grosvenor square. C. 1885-6.

1851 *CADGE, WILLIAM, Surgeon to the Norfolk and Norwich Hospital; 49, St. Giles's street, Norwich. *Trans.* 1.

1885 CAHILL, JOHN, 26, Albert Gate, Hyde park.

1875 CARTER, CHARLES HENRY, M.D., Physician to the Hospital for Women, Soho square; 45, Great Cumberland place, Hyde park.

1853 CARTER, ROBERT BRUDENELL, Ophthalmic Surgeon to, and Lecturer on Ophthalmic Surgery at, St. George's Hospital; 27, Queen Anne street, Cavendish square. *Trans.* 1.

1845 †CARTWRIGHT, SAMUEL, late Professor of Dental Surgery at King's College, London, and Surgeon-Dentist to King's College Hospital; Consulting Surgeon to the Dental Hospital; 32, Old Burlington street. C. 1860-1. *Sci. Com.* 1863.

1879 CARTWRIGHT, S. HAMILTON, Professor of Dental Surgery at King's College, London, and Surgeon Dentist to King's College Hospital; 32, Old Burlington street.

1868 CAVAFY, JOHN, M.D., Physician to St. George's Hospital; 2, Upper Berkeley street, Portman square. *Trans.* 1.

1871 CAYLEY, WILLIAM, M.D., Physician to, and Lecturer on the Principles and Practice of Medicine at, the Middlesex Hospital; Physician to the London Fever Hospital and to the North-Eastern Hospital for Children; 27, Wimpole street, Cavendish square. *Referee*, 1886. *Lib. Com.* 1886. *Trans.* 2.

Elected

1884 CHAFFEY, WAYLAND CHARLES, M.B., 28, Cedars road, Clapham Common.

1845 †CHALK, WILLIAM OLIVER, 3, Nottingham terrace, York gate, Regent's park. C. 1872-3.

1844 †CHAMBERS, THOMAS KING, M.D., Hon. Physician to H.R.H. the Prince of Wales; Consulting Physician to St. Mary's Hospital and to the Lock Hospital; Shrubs Hill House, Sunningdale. C. 1861. V.P. 1867. L. 1869-72. *Referee*, 1851-60, 1866. *Lib. Com.* 1852, 1868. *Trans.* 1.

1879 CHAMPNEYS, FRANCIS HENRY, M.A., M.B., Obstetric Physician to, and Lecturer on Midwifery at, St. George's Hospital; Examiner in Obstetric Medicine in the University of London; 60, Great Cumberland place. *Lib. Com.* 1885-6. *Trans.* 7.

1859 CHANCE, FRANK, M.D., Burleigh House, Sydenham Hill.

1849 CHAPMAN, FREDERICK, Old Friars, Richmond Green, Surrey.

1885 CHAPMAN, PAUL MORGAN, M.D., Physician to the Hereford General Infirmary, 1, St. John street, Hereford. *Trans.* 1.

1877 CHARLES, T. CRANSTOUN, M.D., Lecturer on Practical Physiology at St. Thomas's Hospital; 9, Albert Mansions, Victoria street, Westminster.

1881 *CHAVASSE, THOMAS FREDERICK, M.D., C.M., Surgeon to the Birmingham General Hospital; 24, Temple Row, Birmingham. *Trans.* 2.

1868 CHEADLE, WALTER BUTLER, M.D., *Secretary*, Physician to, and Lecturer on Medicine at, St. Mary's Hospital; Senior Physician to the Hospital for Sick Children; 19, Portman street, Portman square. S. 1886. *Referee*, 1885.

1879 CHEYNE, WILLIAM WATSON, M.B., Assistant Surgeon to King's College Hospital, and Demonstrator of Surgery in King's College, London; 14, Mandeville place, Manchester square, W. *Lib. Com.* 1886.

1873 *CHISHOLM, EDWIN, M.D., Abergeldie, Ashfield, near Sydney, New South Wales.

Elected

1865 CHOLMELEY, WILLIAM, M.D., Physician to the Great Northern Hospital, and Consulting Physician to the Margaret Street Infirmary for Consumption; 63, Grosvenor street, Grosvenor square. C. 1881-2. *Referee*, 1873-80.

1872 CHRISTIE, THOMAS BEITH, M.D., Medical Superintendent, Royal India Asylum, Ealing.

1866 CHURCH, WILLIAM SELBY, M.D., Physician to, and Lecturer on Clinical Medicine at, St. Bartholomew's Hospital; 130, Harley street, Cavendish square. C. 1885-6. *Referee*, 1874-81.

1860 CLARK, SIR ANDREW, Bart., M.D., LL.D., F.R.S., Physician to, and Lecturer on Clinical Medicine at, the London Hospital; 16, Cavendish square. C. 1875.

1879 CLARK, ANDREW, Assistant Surgeon to, and Lecturer on Practical Surgery at, the Middlesex Hospital; 19, Cavendish place, Cavendish square, W.

1839 †CLARK, FREDERICK LE GROS, F.R.S., Consulting Surgeon to St. Thomas's Hospital; The Thorns, Sevenoaks. S. 1847-9. V.P. 1855-6. *Referee*, 1859-81. *Lib. Com.* 1847. *Trans.* 5.

1882 CLARKE, ERNEST, M.D., B.S., 21, Lee terrace, Blackheath.

1848 †CLARKE, JOHN, M.D., 42, Hertford street, May Fair. C. 1866.

1881 CLARKE, W. BRUCE, M.B., Assistant Surgeon to, and Demonstrator of Anatomy at, St. Bartholomew's Hospital; 46, Harley street, Cavendish square.

1842 †CLAYTON, SIR OSCAR MOORE PASSEY, Extra Surgeon-in-Ordinary to H.R.H. the Prince of Wales, and Surgeon-in-Ordinary to H.R.H. the Duke of Edinburgh; 5, Harley street, Cavendish square. C. 1865.

1879 †CLUTTON, HENRY HUGH, M.A., M.B., Assistant Surgeon to, and Lecturer on Forensic Medicine at, St. Thomas's Hospital; 2, Portland place.

1857 COATES, CHARLES, M.D., Consulting Physician to the Bath General and Royal United Hospitals; 10, Circus, Bath.

Elected

1868 COCKLE, JOHN, M.D., F.L.S., Physician to the Royal Free Hospital; 8, Suffolk street, Pall Mall. *Trans.* 2.

1885 COLLINS, WILLIAM MAUNSELL, M.D., 10, Cadogan place.

1865 COOPER, ALFRED, Consulting Surgeon to the West London Hospital; Surgeon to the Lock Hospital and to St. Mark's Hospital; 9, Henrietta street, Cavendish square.

1868 CORNISH, WILLIAM ROBERT, Surgeon-Major, Madras Army; Sanitary Commissioner for Madras; Secretary to the Inspector-General, Indian Medical Department.

1860 *CORRY, THOMAS CHARLES STEUART, M.D., Ormean Terrace, Belfast.

1864 COULSON, WALTER JOHN, Surgeon to the Lock Hospital, 17, Harley street, Cavendish square.

1860 †COUPER, JOHN, Surgeon to the London Hospital; Assistant Surgeon to the Royal London Ophthalmic Hospital; 80, Grosvenor street. C. 1876. *Referee* 1882-3.

1877 COUPLAND, SIDNEY, M.D., Physician to, and Lecturer on Practical Medicine at, the Middlesex Hospital; 14, Weymouth street, Portland place.

1862 COWELL, GEORGE, Surgeon to, and Lecturer on Surgery at, the Westminster Hospital; Surgeon to the Royal Westminster Ophthalmic Hospital; Surgeon to the Victoria Hospital for Children; 3, Cavendish place, Cavendish square. C. 1882-3.

1841 CRAWFORD, MERVYN ARCHDALL NOTT, M.D., Millwood, Wilbury road, Brighton. C. 1853-4.

1868 CRAWFORD, SIR THOMAS, K.C.B., M.D., Director General, Army Medical Department; 6, Whitehall yard, and 5, St. John's park, Blackheath.

1873 CREIGHTON, CHARLES, M.D., 11, New Cavendish street. *Referee*, 1882-6. *Trans.* 1.

1869 *CRESSWELL, PEARSON R., Dowlais, Merthyr Tydvil.

1874 CRIPPS, WILLIAM HARRISON, Assistant Surgeon to St. Bartholomew's Hospital; 2, Stratford place, Oxford street. *Trans.* 1.

Elected

1832 CROCKER, HENRY RADCLIFFE, M.D., Physician to the Skin Department, University College Hospital; Physician to the East London Hospital for Children; 28, Welbeck street, Cavendish square. *Trans.* 1.

1868 CROFT, JOHN, Surgeon to, and Lecturer on Clinical Surgery at, St. Thomas's Hospital; 48, Brook street, Grosvenor square. C. 1884. *Referee*, 1885-86. *Lib. Com.* 1877-8. *Trans.* 1.

1862 CROMPTON, SAMUEL, M.D., Brookmead, Cranleigh, Surrey.

1837 CROOKES, JOHN FARRAR, 45, Augusta gardens, Folkestone.

1872 CROSSE, THOMAS WILLIAM, Surgeon to the Norfolk and Norwich Hospital; 22, St. Giles's street, Norwich.

1849 *CROWFOOT, WILLIAM EDWARD, Beccles, Suffolk.

1879 CUMBERBATCH, A. ELKIN, Aural Surgeon to St. Bartholomew's Hospital; Aural Surgeon to the Great Northern Hospital; 17, Queen Anne street.

1846 CURLING, HENRY, Consulting Surgeon to the Margate Royal Sea-Bathing Infirmary; Augusta Lodge, Ramsgate, Kent.

1837 †CURLING, THOMAS BLIZARD, F.R.S., Consulting-Surgeon to the London Hospital; 27, Brunswick square, Brighton. S. 1845-6. C. 1850. T. 1854-7. V.P. 1859. P. 1871-2. *Referee*, 1844-6, 1851-3, 1858, 1865-70, 1875-9. *Sci. Com.* 1863. *Lib. Com.* 1839. *Trans.* 13. *Pro.* 1.

1873 CURNOW, JOHN, M.D., Professor of Anatomy in King's College, London, and Physician to King's College Hospital; 3, George street, Hanover square. *Referee*, 1884-6.

1847 CURREY, JOHN EDMUND, M.D., Lismore, County Waterford.

1822 CUSACK, CHRISTOPHER JOHN, Chateau d'Eu, France.

1872 DALBY, SIR WILLIAM BARTLETT, M.B., Aural Surgeon to, and Lecturer on Aural Surgery at, St. George's Hospital; 18, Savile row. *Trans.* 3.

1884 DALLAWAY, DENNIS, Whitgift House, Croydon.

Elected

1877 DARBISHIRE, SAMUEL DUKINFIELD, M.D., Physician to the Radcliffe Infirmary, Oxford; 60, High street, Oxford.

1879 DARWIN, FRANCIS, M.B., F.R.S., The Grove, Huntingdon road, Cambridge.

1848 DAUBENY, HENRY, M.D., San Remo, Italy.

1874 DAVIDSON, ALEXANDER, M.D., Physician to the Liverpool Northern Hospital; 2, Gambier terrace, Liverpool.

1853 DAVIES, ROBERT COKER NASH, Rye, Sussex.

1852 DAVIES, WILLIAM, M.D., 2, Marlborough buildings, Bath.

1876 DAVIES-COLLEY, J. NEVILLE C., M.C., Surgeon to, and Lecturer on Anatomy at, Guy's Hospital; 36, Harley street, Cavendish square. *Trans.* 2.

1878 DAVY, RICHARD, F.R.S. Ed., Surgeon to, and Lecturer on Surgery at, the Westminster Hospital; 33, Welbeck street, Cavendish square. *Trans.* 1.

1882 DAWSON, YELVERTON, M.D., Heathlands, Southbourn-on-Sea, Hants.

1867 DAY, WILLIAM HENRY, M.D., Physician to the Samaritan Free Hospital for Women and Children; 10, Manchester square.

1878 DENT, CLINTON THOMAS, Assistant Surgeon to, and Lecturer on Practical Surgery at, St. George's Hospital; 61, Brook street. *Trans.* 2.

1859 †DICKINSON, WILLIAM HOWSHIP, M.D., Physician to, and Lecturer on Medicine at St. George's Hospital, and Consulting Physician to the Hospital for Sick Children; 9, Chesterfield street, Mayfair. C. 1874-5. *Referee*, 1869-73, 1882-6. *Sci. Com.* 1867-79. *Trans.* 13.

1839 †DIXON, JAMES, Consulting Surgeon to the Royal London Ophthalmic Hospital, Moorfields; Harrow Lands, Dorking. L. 1849-55. V.P. 1857-8. T. 1863-4. C. 1866-7. *Referee*, 1865. *Lib. Com.* 1845-8. *Trans.* 4.

Elected

1862 DOBELL, HORACE B., M.D., Consulting Physician to the Royal Hospital for Diseases of the Chest, City road; Streate place, Bournemouth. *Trans.* 2.

1845 DODD, JOHN.

1879 DONKIN, HORATIO, M.B., Physician to the Westminster Hospital; Physician to the East London Hospital for Children; 60, Upper Berkeley street, Portman square.

1877 DORAN, ALBAN HENRY GRIFFITHS, Assistant Surgeon to the Samaritan Free Hospital; 9, Granville place, Portman square. *Trans.* 1.

1863 DOWN, JOHN LANGDON HAYDON, M.D., Physician to, and Lecturer on Clinical Medicine at, the London Hospital; 81, Harley street, Cavendish square. C. 1880. *Trans.* 2.

1867 DRAGE, CHARLES, M.D., Hatfield, Herts.

1884 DRAGE, LOVELL, Hatfield, Herts.

1879 DREWITT, F. G. DAWTREY, M.D., Assistant Physician to the West London Hospital and to the Victoria Hospital for Children; 52, Brook street, Grosvenor square.

1880 DRURY, CHARLES DENNIS HILL, M.D., Bondgate, Darlington.

1865 DRYSDALE, CHARLES ROBERT, M.D., Physician to the Farringdon Dispensary; Assistant-Physician to the Metropolitan Free Hospital; 23, Sackville street, Piccadilly.

1865 †DUCKWORTH, SIR DYCE, M.D., Physician to, and Lecturer on Clinical Medicine at, St. Bartholomew's Hospital; 11, Grafton street, Bond street. C. 1883-4. *Referee* 1885-6. *Trans.* 1.

1876 DUDLEY, WILLIAM LEWIS, M.D., Physician to the City Dispensary; 149, Cromwell road, South Kensington.

1845 DUFF, GEORGE, M.D., High street, Elgin.

1885 DRUMMOND, DAVID, M.D., 7, Saville Place, Newcastle-on-Tyne.

1874 DUFFIN, ALFRED BAYNARD, M.D., Professor of Pathological Anatomy in King's College, London, and Physician to King's College Hospital; 18, Devonshire street, Portland place.

Elected

1871 DUKE, BENJAMIN, Windmill House, Clapham common.

1871 *DUKES, CLEMENT, M.D., B.S., Physician to Rugby School, and Senior Physician to the Hospital of St. Cross, Rugby; Sunnyside, Rugby, Warwickshire.

1867 DUKES, M. CHARLES, M.D., Wellesley Villa, Wellesley road, Croydon.

1880 DUNBAR, JAMES JOHN MACWHIRTER, M.D., Hedingham House, Clapham common.

1877 DUNCAN, JAMES MATTHEWS, M.D., LL.D., F.R.S., Obstetric Physician to, and Lecturer on Midwifery and Diseases of Women at, St. Bartholomew's Hospital; 71, Brook street, Grosvenor square. *Referee*, 1881-6. *Trans.* 1.

1884 DUNCAN, WILLIAM A., M.D., Assistant Obstetric Physician and Teacher of Operative Midwifery, Middlesex Hospital; 6, Harley street, Cavendish square.

1863 DURHAM, ARTHUR EDWARD, F.L.S., Surgeon to, and Lecturer on Surgery at, Guy's Hospital; 82, Brook street, Grosvenor square. C. 1876-7. *Referee*, 1880-1. *Sci. Com.* 1867. *Lib. Com.* 1872-5. *Trans.* 5.

1874 DURHAM, FREDERIC, M.B., 82, Brook street, Grosvenor square.

1843 DURRANT, CHRISTOPHER MERCER, M.D., Consulting Physician to the East Suffolk and Ipswich Hospital; Northgate street, Ipswich, Suffolk.

1872 EAGER, REGINALD, M.D., Northwoods, near Bristol.

1868 EASTES, GEORGE, M.B.Lond., 69, Connaught street, Hyde park square.

1883 EDMUNDS, WALTER, M.C., 79, Lambeth Palace road, Albert Embankment. *Trans.* 2.

1883 EDWARDES, EDWARD JOSHUA, M.D., 17, Orchard street, Portman Square, W.

1884 EDWARDS, FREDERICK SWINFORD, Surgeon to the West London Hospital; 93, Wimpole street, Cavendish square.

Elected

1824 EDWARDS, GEORGE.

1869 ELAM, CHARLES, M.D., 75, Harley street, Cavendish square. *Lib. Com.* 1886.

1848 ELLIS, GEORGE VINER, late Professor of Anatomy in University College, London; Minsterworth, Gloucester. C. 1863-4. *Trans.* 2.

1868 ELLIS, JAMES, M.D., the Sanatorium, Anaheim, Los Angeles County, California.

1854 *ELLISON, JAMES, M.D., Surgeon-in-Ordinary to the Royal Household, Windsor; 14, High street, Windsor.

1842 †ERICHSEN, JOHN ERIC, LL.D.,F.R.S., Surgeon Extraordinary to H.M. the Queen; Emeritus Professor of Surgery in University College, London, and Consulting Surgeon to University College Hospital; 6, Cavendish place, Cavendish sq. C.1855-6. V.P. 1868. P.1879-80. *Referee*, 1866-7, 1884-6. *Lib. Com.* 1844-7, 1854. *Trans.* 2.

1879 EVE, FREDERIC S., Pathological Curator of the Museum, Royal College of Surgeons; Assistant Surgeon to the London Hospital; 15, Finsbury circus. *Trans.* 2.

1877 EWART, WILLIAM, M.D., Assistant Physician to, and Lecturer on Physiology at, St. George's Hospital; 33, Curzon street, Mayfair.

1875 *FAGAN, JOHN, Surgeon to, and Lecturer on Clinical Surgery at, the Belfast Royal Hospital; 1, Glengall place, Belfast.

1869 FAIRBANK, FREDERICK ROYSTON, M.D., 46, Hallgate, Doncaster.

1862 FARQUHARSON, ROBERT, M.D., LL.D., M.P., Migvie Lodge, Porchester gardens, Hyde park; Finzean, Aboyne, Aberdeenshire, and the Reform Club, Pall Mall. *Lib. Com.* 1876-80.

1844 †FARRE, ARTHUR, M.D., F.R.S., Physician Extraordinary to H.M. the Queen; Physician-Accoucheur to H.R.H. the Princess of Wales; 18, Albert Mansions, Victoria street, Westminster. C. 1857. V.P. 1864. *Referee*, 1848-54, 1861-3, 1865-6. *Sci. Com.* 1863. *Lib. Com.* 1847.

Elected

1872 FAYRER, SIR JOSEPH, K.C.S.I., M.D., F.R.S., Honorary Physician to H.M. the Queen, and to H.R.H. the Prince of Wales, and Physician to H.R.H. the Duke of Edinburgh; late Surgeon-General Bengal Medical Service; Examining Medical Officer to the Secretary of State for India in Council; President of the Indian Medical Board; 53, Wimpole street, Cavendish square. *Referee*, 1881-6.

1872 *FENWICK, JOHN C. J., M.D., Physician to the Durham County Hospital; 25, North road, Durham.

1863 FENWICK, SAMUEL, M.D., Physician to the London Hospital; 29, Harley street, Cavendish square. C. 1880. *Referee*, 1882-6. *Trans.* 4.

1880 FERRIER, DAVID, M.D., LL.D., F.R.S., Professor of Forensic Medicine in King's College, London, and Physician to King's College Hospital; Physician for Out-patients to the National Hospital for the Paralysed and Epileptic; 34, Cavendish square. *Trans.* 2.

1852 *FIELD, ALFRED GEORGE.

1849 †FINCHAM, GEORGE TUPMAN, M.D., Consulting Physician to the Westminster Hospital; 13, Belgrave road, Pimlico. C. 1871.

1879 FINLAY, DAVID WHITE, M.D., Physician to, and Lecturer on Forensic Medicine at, the Middlesex Hospital; Physician to the Royal Hospital for Diseases of the Chest; 9, Lower Berkeley street, Portman square.

1866 FISH, JOHN CROCKETT, B.A., M.D., 92, Wimpole street, Cavendish square.

1866 FITZ-PATRICK, THOMAS, M.D., M.A., 30, Sussex gardens, Hyde park.

1842 FLETCHER, THOMAS BELL ELCOCK, M.D., Consulting Physician to the Birmingham General Hospital; 8, Clarendon crescent, Leamington. *Trans.* 1.

1864 *FOLKER, WILLIAM HENRY, Surgeon to the North Staffordshire Infirmary; Bedford House, Hanley, Staffordshire.

1877 DE FONMARTIN, HENRY, M.D., Parkhurst, Isle of Wight.

Elected

1848 †Forbes, John Gregory, Egerton House, Egerton, Ashford, Kent. C. 1868-9. *Lib. Com.* 1855. *Trans.* 2.

1865 Foster, Sir Balthazar Walter, M.D., Professor of Medicine at the Queen's College, Birmingham, and Physician to the Birmingham General Hospital; 14, Temple row, Birmingham.

1883 Fowler, James Kingston, M.A., M.D., Assistant Physician to, and Lecturer on Pathological Anatomy at, the Middlesex Hospital, and Assistant Physician to th Hospital for Consumption, Brompton; 35, Clarges street, Piccadilly.

1859 Fox, Edward Long, M.D., Consulting Physician to the Bristol Royal Infirmary; Church House, Clifton, Gloucestershire.

1880 Fox, Thomas Colcott, B.A., M.B., Physician to the Skin Department of the Paddington Green Hospital for Children, and Assistant Physician to the Victoria Hospital for Children; 14, Harley street, Cavendish square. *Trans.* 1.

1858 Fox, Wilson, M.D., F.R.S., *Librarian*, Physician-Extraordinary to H.M. the Queen; Physician in Ordinary to the Duke and Duchess of Edinburgh; Holme Professor of Clinical Medicine in University College, London, and Physician to University College Hospital; 67, Grosvenor street. C. 1875-6. L. 1883-6. *Referee*, 1869-74. *Lib. Com.* 1866-70, 1874. *Trans.* 3.

1871 Frank, Philip, M.D., Cannes, France.

1884 *Franks, Kendal, M.D., Surgeon to the Adelaide Hospital and to the Throat and Ear Hospital, Dublin; 69, Fitzwilliam square, Dublin.

1843 Fraser, Patrick, M.D. C. 1866.

1868 Freeman, William Henry, 21, St. George's square, South Belgravia.

1836 †French, John George, 10, Cunningham place, St. John's Wood road. C. 1852-3.

Elected

1884 FULLER, CHARLES CHINNER, 10, St. Andrew's place, Regent's park.

1883 FULLER, HENRY ROXBURGH, M.D., 45, Curzon street, May Fair.

1876 FURNER, WILLOUGHBY, Assistant Surgeon to the Sussex County Hospital; 2, Brunswick place, Brighton.

1864 *GAIRDNER, WILLIAM TENNANT, M.D., LL.D., Physician in Ordinary to H.M. the Queen in Scotland; Professor of the Practice of Medicine in the University of Glasgow; Physician to the Western Infirmary, Glasgow; 225, St. Vincent street, Glasgow.

1874 GALABIN, ALFRED LEWIS, M.A., M.D., Obstetric Physician to, and Lecturer on Midwifery and the Diseases of Women at, Guy's Hospital; Assistant Physician to the Hospital for Sick Children; 49, Wimpole street, Cavendish square. *Referee*, 1882-6. *Lib. Com.* 1883-4. *Trans.* 2.

1883 GALTON, JOHN CHARLES, M.A., F.L.S., 45, Great Marlborough street.

1885 GAMGEE, ARTHUR, M.D., F.R.S., Fullerian Professor of Physiology in the Royal Institution of Great Britain; 11, Warrior square, St. Leonard's-on-sea.

1865 GANT, FREDERICK JAMES, Senior Surgeon to the Royal Free Hospital; 16, Connaught square, Hyde park. C. 1880-81. *Referee*, 1886. *Lib. Com.* 1882-5. *Trans.* 3.

1867 GARLAND, EDWARD CHARLES, Yeovil, Somerset.

1867 GARLIKE, THOMAS W., Malvern Cottage, Churchfield road, Ealing.

1854 †GARROD, ALFRED BARING, M.D., F.R.S., Consulting Physician to King's College Hospital; 10, Harley street, Cavendish square. C. 1867. V.P. 1880-81. *Referee*, 1855-65. *Trans.* 8.

1879 GARSTANG, THOMAS WALTER HARROPP, The Heath, Knutsford, Cheshire.

Elected

1851 †GASKOIN, GEORGE, Surgeon to the British Hospital for Diseases of the Skin; The Priory, Caerleon, Monmouthshire. C. 1875-6. *Trans.* 2.

1819 GAULTER, HENRY.

1866 GEE, SAMUEL JONES, M.D., Physician to, and Lecturer on Medicine at, St. Bartholomew's Hospital; Consulting Physician to the Hospital for Sick Children; 54, Wimpole street, Cavendish square. C. 1883-4. *Sci. Com.* 1879. *Referee*, 1885-6. *Lib. Com.* 1871-6. *Trans.* 1.

1885 GELL, HENRY WILLINGHAM, Balliol College, Oxford.

1878 GERVIS, HENRY, M.D., Obstetric Physician to, and Lecturer on Obstetric Medicine at, St. Thomas's Hospital; 40, Harley street, Cavendish square. *Referee*, 1884-6.

1884 GIBBES, HENEAGE, M.D., Physician to the Metropolitan Dispensary; Lecturer on Morbid Histology, Westminster Hospital; 44, Charleville road, West Kensington.

1880 GIBBONS, ROBERT ALEXANDER, M.D., Physician to the Grosvenor Hospital for Women and Children; 32, Cadogan place.

1877 GODLEE, RICKMAN JOHN, Surgeon to University College Hospital, and Teacher of Operative Surgery in University College, London; Surgeon to the North-Eastern Hospital for Children, and to the Hospital for Consumption, Brompton; 81, Wimpole street, Cavendish square. *Referee*, 1886. *Trans.* 2.

1870 GODSON, CLEMENT, M.D., Assistant-Physician-Accoucheur to St. Bartholomew's Hospital; Consulting Physician to the City of London Lying-in Hospital; 9, Grosvenor street, Grosvenor square.

1886 GOLDING-BIRD, CUTHBERT HILTON, M.B., Assistant Surgeon and Lecturer on Physiology at Guy's Hospital; 13, St. Thomas street, Southwark.

1851 GOODFELLOW, STEPHEN JENNINGS, M.D., Consulting Physician to the Middlesex Hospital; Swinnerton Lodge, near Dartmouth, Devon. C. 1864-5. *Referee*, 1860-3. *Lib. Com.* 1863. *Trans.* 2.

Elected

1883 GOODHART, JAMES FREDERIC, M.D., Assistant Physician to, and Curator of the Museum at, Guy's Hospital; Physician to the Evelina Hospital for Sick Children; 25, Weymouth street, Portland place.

1877 GOULD, ALFRED PEARCE, M.S., Assistant Surgeon to the Middlesex Hospital; 16, Queen Anne street, Cavendish square. *Trans.* 1.

1873 GOWERS, WILLIAM RICHARD, M.D., Assistant Professor of Clinical Medicine in University College, London, and Physician to University College Hospital; Physician for Out-patients to the National Hospital for the Paralysed and Epileptic; 50, Queen Anne street, Cavendish square. *Lib. Com.* 1884-6. *Trans.* 6.

1851 †GOWLLAND, PETER YEAMES, Surgeon to St. Mark's Hospital; Surgeon-Major Hon. Artillery Company; 34, Finsbury square.

1846 GREAM, GEORGE THOMPSON, M.D., Physician-Accoucheur to H.R.H. the Princess of Wales; Mixbury, Eastbourne, Sussex. C. 1863.

1868 GREEN, T. HENRY, M.D., Physician to, and Lecturer on Pathology at, Charing Cross Hospital; Assistant-Physician to the Hospital for Consumption, Brompton; 74, Wimpole street, Cavendish square. C. 1886. *Referee*, 1882-5.

1875 *GREENFIELD, WILLIAM SMITH, M.D., Professor of General Pathology in the University of Edinburgh; 7, Heriot row, Edinburgh. *Sci. Com.* 1879. *Referee*, 1881.

1843 †GREENHALGH, ROBERT, M.D., Consulting Physician to the Samaritan Free Hospital for Women and Children, and to the City of London Lying-in Hospital; 35, Cavendish square. C. 1871-2. *Referee*, 1876-7. *Trans.* 1.

1860 GREENHOW, EDWARD HEADLAM, M.D., F.R.S., Consulting Physician to the Middlesex Hospital; and Consulting Physician to the Western General Dispensary; Castle Lodge, Reigate. C. 1876-7. *Referee*, 1870-5. *Trans.* 3.

Elected

1882 GRESSWELL, DAN ASTLEY, M.B., 87, Queen's crescent, Haverstock hill.

1885 GRIFFITH, WALTER SPENCER ANDERSON, M.B., Physician to the Samaritan Free Hospital for Women and Children; 114, Harley street, Cavendish square.

1884 GRIFFITHS, HERBERT TYRRELL, M.D., 57, Brook street.

1868 GRIGG, WILLIAM CHAPMAN, M.D., Assistant Obstetric Physician to the Westminster Hospital; Physician to the In-Patients, Queen Charlotte's Lying-in-Hospital; 27, Curzon street, Mayfair.

1852 GROVE, JOHN, Fyning, Austen road, Guildford.

1860 GUENEAU DE MUSSY, HENRI, M.D.; 15, Rue du Cirque, Paris. *Lib. Com.* 1863-5.

1849 †GULL, SIR WILLIAM WITHEY, Bart., M.D., D.C.L., LL.D., F.R.S., Physician-Extraordinary to H.M. the Queen; and Physician in Ordinary to H.R.H. the Prince of Wales; Member of the Senate of the University of London; Consulting Physician to Guy's Hospital; 74, Brook street, Grosvenor square. C. 1864. V.P. 1874. *Referee*, 1855-63. *Trans.* 4.

1885 GULLIVER, GEORGE, M.B., Assistant Physician to, and Lecturer on Comparative Anatomy at, St. Thomas's Hospital; 16, Welbeck street.

1883 GUNN, ROBERT MARCUS, M.B., Assistant Surgeon to the Royal London Ophthalmic Hospital, Moorfields; 54, Queen Anne street, Cavendish square.

1854 †HABERSHON, SAMUEL OSBORNE, M.D., 70, Brook street, Grosvenor square. S. 1867. C. 1869-70. V.P. 1881-2. *Referee*, 1862-6, 1868, 1871-80. *Trans.* 3.

1885 HAIG, ALEXANDER, M.B., Casualty Physician to St. Bartholomew's Hospital; 30, Welbeck street, Cavendish square.

1881 HALL, FRANCIS DE HAVILLAND, M.D., Assistant Physician, and Physician to the Throat Department, and Lecturer on Forensic Medicine at the Westminster Hospital; Physician to St. Mark's Hospital; 47, Wimpole street, Cavendish square.

Elected

1885 HALLIBURTON, WILLIAM DOBINSON, M.D., Assistant Professor of Physiology, University College, London; 135, Gower street.

1870 HAMILTON, ROBERT, Surgeon to the Royal Southern Hospital, Liverpool; 1 Prince's road, Liverpool.

1874 HARDIE, GORDON KENMURE, M.D., Deputy Inspector General of Hospitals; Florence road, Ealing, and Duff House, Banff, N.B.

1856 †HARE, CHARLES JOHN, M.D., late Professor of Clinical Medicine in University College, London, and late Physician to University College Hospital; Berkeley House, 15, Manchester square. C. 1873-4.

1857 HARLEY, GEORGE, M.D., F.R.S. 25, Harley street, Cavendish square. C. 1871-2. *Referee*, 1865-70, 1873-6. *Sci. Com.* 1862-3. *Trans.* 1.

1864 HARLEY, JOHN, M.D., F.L.S., Physician to, and Lecturer on General Anatomy and Physiology at, St. Thomas's Hospital; 39, Brook street, Grosvenor square. S. 1875-7. C. 1879-80. *Referee*, 1871-4, 1882-6. *Sci. Com.* 1879. *Trans.* 10.

1880 HARRIS, VINCENT DORMER, M.D., Assistant Physician to the Victoria Park Hospital; Demonstrator of Physiology at St. Bartholomew's Hospital; 31, Wimpole street, Cavendish square.

1870 HARRISON, REGINALD, Surgeon to the Liverpool Royal Infirmary, and Lecturer on Clinical Surgery in the Victoria University; 41, Rodney street, Liverpool. *Trans.* 1.

1854 HAVILAND, ALFRED.

1870 HAWARD, J. WARRINGTON, Surgeon to, and Lecturer on Clinical Surgery at, St. George's Hospital; 16, Savile row, Burlington gardens. C. 1885. *Lib. Com.* 1881–4. *Trans.* 1.

1838 †HAWKINS, CHARLES, Inspector of Anatomical Schools in London; 9, Duke street, Portland place. C. 1846-7. S. 1850. V.P. 1858. T. 1861-2. *Referee*, 1859-60. *Lib. Com.* 1843. *Trans.* 2.

Elected

1885 HAWKINS, FRANCIS HENRY, M.B., Physician to St. George's and St. James's Dispensary; 22, Henrietta street, Cavendish square.

1848 †HAWKSLEY, THOMAS, M.D., Consulting Physician to the Margaret street Dispensary for Consumption and Diseases of the Chest; 11, Albert Mansions, Victoria street, and Beomands, Chertsey, Surrey.

1875 HAYES, THOMAS CRAWFORD, M.D., Physician-Accoucheur and Physician for Diseases of Women and Children to King's College Hospital; 17, Clarges street, Piccadilly.

1860 HAYWARD, HENRY HOWARD, Surgeon Dentist to, and Lecturer on Dental Surgery at, St. Mary's Hospital; 38, Harley street, Cavendish square. C. 1878-9.

1861 HAYWARD, WILLIAM HENRY, Corby, Grantham.

1848 *HEALE, JAMES NEWTON, M.D.

1865 HEATH, CHRISTOPHER, Holme Professor of Clinical Surgery in University College, London; and Surgeon to University College Hospital; 36, Cavendish square. C. 1880. *Lib. Com.* 1870-3. *Trans.* 3.

1850 HEATON, GEORGE, M.D., Boston, U.S.

1882 HENSLEY, PHILIP JOHN., M.D., Assistant Physician and Lecturer on Forensic Medicine to St. Bartholomew's Hospital; 4, Henrietta street, Cavendish square.

1821 HERBERSKI, VINCENT, M.D., Professor of Medicine in the University of Wilna.

1877 HERMAN, GEORGE ERNEST, M.B., Obstetric Physician to, and Lecturer on Midwifery at, the London Hospital; 7, West street, Finsbury circus. *Trans.* 1.

1877 HERON, GEORGE ALLAN, M.D., Physician to the City of London Hospital for Diseases of the Chest, Victoria Park; 57, Harley street, Cavendish square.

1883 HERRINGHAM, WILMOT PARKER, M.B., 22, Bedford square.

Elected

1843 †Hewett, Sir Prescott Gardner, Bart., F.R.S., Serjeant-Surgeon to H.M. the Queen; Surgeon in Ordinary to H.R.H. the Prince of Wales; Consulting Surgeon to St. George's Hospital; Corresponding Member of the "Académie de Médecine," and of the "Société de Chirurgie," Paris; Chesnut Lodge, Horsham, Sussex. C. 1859. V.P. 1866-7. *Referee*, 1850-8, 1860-5, 1868-83. *Sci. Com.* 1863. *Lib. Com.* 1846-7. *Trans.* 7.

1855 Hewitt, W. M. Graily, M.D., Professor of Midwifery in University College, London, and Obstetric Physician to University College Hospital; 36, Berkeley square. C. 1876. *Referee*, 1868-75, 1877-86. *Lib. Com.* 1868, 1874.

1880 Hicks, Charles Cyril, M.D., Wokingham, Berks.

1873 Higgens, Charles, Assistant Ophthalmic Surgeon to, and Lecturer on Ophthalmic Surgery at, Guy's Hospital; 38, Brook street, Grosvenor square. *Trans.* 2.

1862 Hill, M. Berkeley, M.B., *Vice-President*, Professor of Clinical Surgery in University College, London, and Surgeon to University College Hospital; Surgeon to the Lock Hospital; 66, Wimpole street, Cavendish square. C. 1878-9. S. 1881-4. V.P. 1885-6. *Trans.* 1.

1867 Hill, Samuel, M.D., 22, Mecklenburgh square.

1861 *Hoffmeister, Sir William Carter, M.D., Surgeon to H.M. the Queen in the Isle of Wight; Clifton House, Cowes, Isle of Wight.

1843 †Holden, Luther, Consulting Surgeon to St. Bartholomew's Hospital, to the Metropolitan Dispensary, and to the Foundling Hospital; Pinetoft, Ipswich. C. 1859. L. 1865. V.P. 1874. *Referee*, 1866-7. *Lib. Com.* 1858.

1879 Holland, Philip Alexander, M.A.

1868 Hollis, William Ainslie, M.A., M.D., Assistant-Physician to the Sussex County Hospital; 8, Cambridge road, Brighton.

Elected

1861 HOLMAN, WILLIAM HENRY, M.B., 68, Adelaide road, South Hampstead.

1856 HOLMES, TIMOTHY, M.A., *Treasurer*, Surgeon to St. George's Hospital; Corresponding Member of the "Société de Chirurgie," Paris; 18, Great Cumberland place, Hyde park. C. 1869-70. L. 1873-7. S. 1878-80. V.P. 1881-2. T. 1885-6. *Referee*, 1866-8, 1872, 1883-4. *Sci. Com.* 1867. *Lib. Com.* 1863-5. *Trans.* 8.

1846 †HOLT, BARNARD WIGHT, Consulting Surgeon to the Westminster Hospital; Medical Officer of Health for Westminster, 14, Savile row, Burlington gardens. C. 1862-3. V.P. 1879-80.

1846 †HOLTHOUSE, CARSTEN, 35, Essex street, Strand. C. 1863. *Referee* 1870-6. *Lib Com.* 1859-60.

1878 HOOD, DONALD WILLIAM CHARLES, M.D., Senior Physician to the North-West London Hospital; Physician to the West London Hospital; 43, Green street, Park lane.

1883 HORSLEY, VICTOR ALEXANDER HADEN, F.R.S., Assistant Surgeon to University College Hospital, and Assistant Professor of Pathological Anatomy in University College, London; Superintendent of the Brown Institution, Wandsworth road; 80, Park street, Grosvenor Square.

1878 HOUGHTON, WALTER B., M.D., Church Villa, Warrior square, St. Leonards-on-Sea.

1865 HOWARD, BENJAMIN, M.D., New York, U.S.

1881 HOWARD, HENRY, M.B., abroad [6, The Terrace, Mount Pleasant, Cambridge].

1874 HOWSE, HENRY GREENWAY, M.S., Surgeon to, and Lecturer on Anatomy at, Guy's Hospital; Surgeon to the Evelina Hospital for Sick Children; 10, St. Thomas's street, Southwark. *Sci. Com.* 1879. *Trans.* 2.

1886 HUDSON, CHARLES LEOPOLD, Middlesex Hospital.

1884 HUGGARD, WILLIAM R., M.D. [Place de la Synagogue, 2, Genève.]

Elected

1857 HULKE, JOHN WHITAKER, F.R.S., *Librarian*, Surgeon to the Middlesex Hospital; Surgeon to the Royal London Ophthalmic Hospital, Moorfields; 10, Old Burlington street. C. 1871-2. S. 1876-7. L. 1879-86. *Sci. Com.* 1867. *Lib. Com.* 1864-8. *Trans.* 8.

1844 †HUMBY, EDWIN, M.D., 83, Hamilton terrace, St. John's wood. C. 1866-7.

1855 HUMPHRY, GEORGE MURRAY, M.D., F.R.S., Surgeon to Addenbrooke's Hospital; Professor of Surgery in the University of Cambridge. *Trans.* 6.

1882 HUMPHRY, LAURENCE, M.B., 3, Trinity street, Cambridge.

1873 HUNTER, SIR W. GUYER, M.D., M.P., Hon. Surgeon to H.M. the Queen; late Principal of, and Professor of Medicine in, Grant Medical College, Bombay; Surgeon-General Bombay Army; 21, Norfolk crescent, Hyde park.

1849 HUSSEY, EDWARD LAW, Consulting Surgeon to the County Lunatic Asylum and the Warneford Asylum; 24, Winchester Road, Oxford. *Trans.* 1.

1856 HUTCHINSON, JONATHAN, F.R.S., Consulting Surgeon to, and Emeritus Professor of Surgery at, the London Hospital; Consulting Surgeon to the Royal London Ophthalmic Hospital, Moorfields; and Surgeon to the Hospital for Diseases of the Skin; 15, Cavendish square. C. 1870. V.P. 1882. *Referee*, 1876-81, 1883-6. *Lib. Com.* 1864-5. *Trans.* 13. *Pro.* 2.

1820 HUTCHINSON, WILLIAM, M.D.

1840 †HUTTON, CHARLES, M.D., 26, Lowndes street, Belgrave square. C. 1858-9.

1847 IMAGE, WILLIAM EDMUND, Herringswell House, Mildenhall, Suffolk. *Trans.* 1.

1856 INGLIS, CORNELIUS, M.D., Cairo. [Athenæum Club, Pall Mall.]

1871 JACKSON, J. HUGHLINGS, M.D., F.R.S., Physician to the London Hospital; Physician to the National Hospital for the Paralysed and Epileptic; 3, Manchester square.

Elected

1841 †JACKSON, PAUL, 51, Wellington road, St. John's Wood. C. 1862.

1863 JACKSON, THOMAS VINCENT, Senior Surgeon to the Wolverhampton and Staffordshire General Hospital; 47, Waterloo road, south, Wolverhampton.

1883 JACOBSON, WALTER HAMILTON ACLAND, B.A., M.B., Assistant Surgeon to Guy's Hospital; Surgeon to the Royal Hospital for Children and Women; 41, Finsbury square. *Trans.* 1.

1825 JAMES, JOHN B., M.D.

1883 *JENKINS, EDWARD JOHNSTONE, M.D., The Australian Club, Sydney, New South Wales.

1851 †JENNER, SIR WILLIAM, Bart., M.D., K.C.B., D.C.L., LL.D., F.R.S., Physician in Ordinary to H.M. the Queen, and to H.R.H. the Prince of Wales; Emeritus Professor of Clinical Medicine in University College, London; and Consulting Physician to University College Hospital; Member of the Senate of the University of London; 63, Brook street, Grosvenor square. C. 1864. V.P. 1875. *Referee*, 1855, 1859-63. *Trans.* 3.

1884 JENNINGS, CHARLES EGERTON, M.S., M.B., 75, Park street, Grosvenor square.

1881 JENNINGS, WILLIAM OSCAR, M.D., 8, Rue Roy, Paris.

1884 JESSETT, FREDERIC BOWREMAN, Surgeon to the Royal General Dispensary; 16, Upper Wimpole street.

1883 JESSOP, WALTER H. H., M.B., Demonstrator of Anatomy at St. Bartholomew's Hospital; 73, Harley street.

1851 JOHNSON, EDMUND CHARLES, Corresponding Member of the Medical and Philosophical Society of Florence, and of "l'Institut Génevois."

1847 †JOHNSON, GEORGE, M.D., F.R.S., Consulting Physician to King's College Hospital; Member of the Senate of the University of London; 11, Savile row, Burlington gardens. C. 1862-3. V.P. 1870. P. 1884-5. L. 1878-80. *Referee*, 1853-61, 1864-9. *Lib. Com.* 1860-1. *Trans.* 10. *Pro.* 1.

Elected

1881 JOHNSON, GEORGE LINDSAY, M.A., M.D., Cortina, Netherhall terrace, South Hampstead, and 14, Stratford place, Oxford street.

1884 JOHNSTON, JAMES, M.D., 40, Brook street, Grosvenor square.

1848 JOHNSTONE, ATHOL ARCHIBALD WOOD, Consulting Surgeon to the Royal Alexandra Hospital for Sick Children, St. Moritz House, 61, Dyke road, Brighton. *Lib. Com.* 1860. *Trans.* 1.

1876 JONES, LESLIE HUDSON, M.D., Limefield House, Cheetham hill, Manchester.

1875 *JONES, PHILIP SYDNEY, M.D., Consulting Surgeon to the Sydney Infirmary; Examiner in Medicine, Sydney University; 10, College street, Sydney, New South Wales. [Agents: Messrs. D. Jones & Co., 1, Gresham buildings, Basinghall street.]

1859 JONES, WILLIAM PRICE, M.D., Claremont road, Surbiton, Kingston.

1865 JORDAN, FURNEAUX, Surgeon to the Queen's Hospital, and Professor of Surgery at the Queen's College, Birmingham; Gate House, Edmund street, Birmingham.

1881 JULER, HENRY EDWARD, Assistant Surgeon Royal Westminster Ophthalmic Hospital; Junior Ophthalmic Surgeon to St. Mary's Hospital; 77, Wimpole street, Cavendish square.

1816 *KAUFFMANN, GEORGE HERMANN, M.D., Hanover.

1882 KEETLEY, CHARLES R. B., Senior Surgeon to the West London Hospital; Surgeon to the Surgical Aid Society; 10, George street, Hanover square.

1872 KELLY, CHARLES, M.D., Professor of Hygiene in King's College, London, and Medical Officer of Health for the West Sussex Combined Sanitary District; Broadwater road, Worthing, Sussex.

1848 *KENDELL, DANIEL BURTON, M.D., Heath House, Wakefield, Yorkshire.

Elected

1884 KESER, JEAN SAMUEL, M.D., Surgeon to the French Hospital, Leicester place; 60, Queen Anne street.

1877 *KHORY, RUSTONJEE NASERWANJEE, M.D., Physician to the Parell Dispensary, Bombay; Girgaum road, Bombay.

1857 KIALLMARK, HENRY WALTER, 5, Pembridge gardens, Bayswater.

1881 KIDD, PERCY, M.A., M D., Assistant Physician to the Hospital for Consumption, Brompton; 60, Brook street, Grosvenor square. *Trans.* 3.

1851 †KINGDON, JOHN ABERNETHY, Surgeon to the City of London Truss Society, and Consulting Surgeon to the City Dispensary; 2, New Bank buildings, Lothbury. C. 1866-7. V.P. 1872-3. *Sci. Com.* 1867. *Trans.* 1.

1885 KLEIN, EDWARD EMANUEL, M.D., F.R.S., Lecturer on Physiology, St. Bartholomew's Hospital; 94, Philbeach gardens, Earl's Court.

1883 KNAPTON, GEORGE, Strathgyle, Portswood, Southampton.

1840 †LANE, SAMUEL ARMSTRONG, Consulting Surgeon to St. Mary's Hospital and to the Lock Hospital; 49, Norfolk square, Hyde park. C. 1849-50. V.P. 1865. *Referee*, 1850.

1884 LANE, WILLIAM ARBUTHNOT, M.S., Assistant Surgeon to the Hospital for Sick Children; 14, St. Thomas's street, Southwark. *Trans.* 1.

1882 LANG, WILLIAM, Ophthalmic Surgeon to, and Lecturer on Ophthalmic Surgery at, the Middlesex Hospital; Assistant Surgeon to the Royal London Ophthalmic Hospital, Moorfields; 26, Upper Wimpole street, Cavendish square.

1865 LANGTON, JOHN, Surgeon to, and Lecturer on Anatomy at, St. Bartholomew's Hospital; Surgeon to the City of London Truss Society; 2, Harley street, Cavendish square. C. 1881-2. *Referee*, 1885-6. *Lib. Com.* 1879-80.

Elected

1873 *Larcher, O., M.D., Laureate of the Institute of France, of the Medical Faculty, and Academy of Paris, &c.; 97, Rue de Passy, Passy, Paris.

1862 Latham, Peter Wallwork, M.A., M.D., Downing Professor of Medicine, Cambridge University; Physician to Addenbrooke's Hospital, Cambridge; 17, Trumpington street, Cambridge.

1816 Lawrence, G. E.

1884 Lawson, George, Surgeon-Oculist to H.M. the Queen; Surgeon to the Royal London Ophthalmic Hospital and to the Middlesex Hospital; 12, Harley street, Cavendish square.

1880 Laycock, George Lockwood, M.B., Physician to the Paddington Green Children's Hospital; 12, Upper Berkeley street, Portman square.

1882 Ledwich, Edward l'Estrange, Lecturer on Surgical and Descriptive Anatomy in the Ledwich School of Medicine, Dublin; 23, Upper Leeson Street, Dublin.

1843 †Lee, Henry, Consulting Surgeon to St. George's Hospital; 9, Savile row, Burlington gardens. C. 1856-7. L. 1863-4. V.P. 1868-9. *Referee*, 1855, 1866-8. *Sci. Com.* 1867. *Trans.* 14. *Pro.* 2.

1884 Lee, Robert James, M.D., Assistant Physician to the Hospital for Sick Children; 6, Savile row.

1883 Leeson, John Rudd, M.D., C.M., 6, Clifden road, Twickenham.

1869 Legg, John Wickham, M.D., Assistant Physician to, and Lecturer on Pathological Anatomy at, St. Bartholomew's Hospital; 47, Green street, Park lane. C. 1886. *Referee*, 1882-5. *Lib. Com.* 1878-85. *Trans.* 2.

1836 Leighton, Frederick, M.D.

1872 Liebreich, Richard, Consulting Ophthalmic Surgeon to St. Thomas's Hospital; Paris.

1878 Lister, Sir Joseph, Bart., D.C.L., LL.D., F.R.S., Surgeon Extraordinary to H.M. the Queen; Professor of Clinical Surgery at King's College, London; and Surgeon to King's College Hospital; 12, Park crescent, Regent's park.

Elected

1872 *LITTLE, DAVID, M.D., Senior Surgeon to the Royal Eye Hospital, Manchester; 21, St. John street, Manchester.

1871 LITTLE, LOUIS STROMEYER, Shanghai, China.

1819 LLOYD, ROBERT, M.D.

1820 LOCHER, J. G., M.C.D., Town Physician of Zurich. *Trans.* 2.

1881 LOCKWOOD, CHARLES BARRETT, Surgeon to the Great Northern Central Hospital, and Demonstrator of Anatomy and Operative Surgery at St. Bartholomew's Hospital; 19, Upper Berkeley street. *Trans.* 1.

1860 LONGMORE, SIR THOMAS, C.B., Hon. Surgeon to H.M. the Queen; Surgeon-General, Army Medical Staff, and Professor of Military Surgery, Army Medical School, Netley, Southampton; Woolston Lawn, Woolston, Hants. *Trans.* 2.

1836 LÖWENFELD, JOSEPH S., M.D., Berbice.

1871 LOWNDS, THOMAS MACKFORD, M.D., late Professor of Anatomy and Physiology at Grant Medical College, Bombay; Egham Hill, Surrey.

1881 LUCAS, RICHARD CLEMENT, Senior Assistant Surgeon to, and Demonstrator of Operative and Practical Surgery at, Guy's Hospital; Surgeon to the Evelina Hospital for Sick Children; 18, Finsbury square.

1883 LUND, EDWARD, Professor of Surgery, and Member of Senate, Victoria University, Manchester; Consulting Surgeon to the Manchester Royal Infirmary; 22, St. John street, Manchester.

1857 LYON, FELIX WILLIAM, M.D., 7, South Charlotte street, Edinburgh.

1867 MABERLY, GEORGE FREDERICK, Mailai Valley, Nelson, New Zealand.

1873 MACCARTHY, JEREMIAH, M.A., Surgeon to the London Hospital and Lecturer on Physiology at the London Hospital Medical College; 15, Finsbury square. C. 1886. *Lib. Com.* 1882-5.

Elected

1867 MAC CORMAC, SIR WILLIAM, M.A., Surgeon to, and Lecturer on Surgery at, St. Thomas's Hospital; 13, Harley street. C. 1884-5. *Trans.* 1.

1862 *M'DONNELL, ROBERT, M.D., F.R.S., Surgeon to Steevens' Jervis street Hospitals; 89, Merrion square west, Dublin. *Trans.* 2.

1880 *MACFARLANE, ALEXANDER WILLIAM, M.D., Consulting Physician to the Kilmarnock Fever Hospital and Infirmary, and Examiner in Medicine and Clinical Medicine, University of Glasgow; Walmer, Kilmarnock, N.B.

1866 MACGOWAN, ALEXANDER THORBURN, M.D., Vyvyan House, Clifton, near Bristol.

1880 MCHARDY, MALCOLM MACDONALD, Ophthalmic Surgeon to King's College Hospital, and Professor of Ophthalmic Surgery in King's College, London; Surgeon to the Royal South London Ophthalmic Hospital; 5, Savile row.

1822 MACINTOSH, RICHARD, M.D.

1859 *M'INTYRE, JOHN, M.D., Odiham, Hants.

1873 MACKELLAR, ALEXANDER OBERLIN, M.S.I., Surgeon to St. Thomas's Hospital; Surgeon-in-Chief to the Metropolitan Police Force; 22, George street, Hanover square.

1881 MACKENZIE, STEPHEN, M.D., Physician to the London Hospital, and Lecturer on the Principles and Practice of Medicine at the London Hospital Medical College; Physician to the Royal London Ophthalmic Hospital; 26, Finsbury square. *Trans.* 1.

1885 MACKERN, JOHN, M.D., Assistant Physician to the Chelsea Hospital for Women; 30, Cambridge street, Hyde park.

1876 MACKEY, EDWARD, M.D., Assistant Physician to the Sussex County Hospital; 1, Brunswick road, Hove, Brighton.

1854 *MACKINDER, DRAPER, M.D., Consulting Surgeon to the Dispensary, Gainsborough, Lincolnshire.

Elected

1879 MACLAGAN, THOMAS JOHN, M.D., Physician-in-Ordinary to their R.H. the Prince and Princess Christian of Schleswig-Holstein ; 9, Cadogan place, Belgrave square.

1876 MACNAMARA, CHARLES N., Surgeon to, and Lecturer on Surgery at, the Westminster Hospital ; Surgeon to the Royal Westminster Ophthalmic Hospital ; Surgeon-Major Bengal Medical Service ; Fellow of the Calcutta University ; 13, Grosvenor street. *Referee*, 1884-6. *Lib. Com.* 1886.

1881 MACREADY, JONATHAN FORSTER CHRISTIAN HORACE, Surgeon to the Great Northern Hospital ; 51, Queen Anne street, Cavendish square.

1880 MADDICK, EDMUND DISTIN, 2, Chandos street, Cavendish square.

1886 MAGUIRE, ROBERT, M.D., Warden of St. Mary's Hospital Residential College ; 33, Westbourne Terrace.

1880 MAKINS, GEORGE HENRY, Assistant Surgeon to the Evelina Hospital for Children ; 2, Queen street, May Fair.

1885 MALCOLM, JOHN DAVID, M.B., Surgeon in charge of Out-Patients, Samaritan Free Hospital ; 24, Bryanston street, Portman square.

1876 MALLAM, BENJAMIN, Rose Bank, Blackall road, Exeter.

1855 MARCET, WILLIAM, M.D., F.R.S. ; 39, Grosvenor street. C. 1871. *Referee*, 1866-70, 1883-6. *Sci. Com.* 1863. *Lib. Com.* 1866-8. *Trans.* 3.

1867 MARSH, F. HOWARD, *Secretary*, Assistant Surgeon to, and Lecturer on Anatomy at, St. Bartholomew's Hospital ; Surgeon to the Hospital for Sick Children, Great Ormond street ; 30, Bruton street, Berkeley square. C. 1882-3. S. 1885-6. *Lib. Com.* 1880-1. *Trans.* 4.

1838 MARSH, THOMAS PARR, M.D.

1851 †MARSHALL, JOHN, F.R.S., Professor of Anatomy to the Royal Academy of Arts ; Emeritus Professor of Surgery in University College, London, and Consulting Surgeon to University College Hospital ; 10, Savile row, Burlington gardens. C. 1866. V.P. 1875-6. P. 1882-3. *Referee*, 1867, 1871-4, 1877-81. *Trans.* 3.

Elected

1884 MARTIN, SIDNEY HARRIS COX, M.D.; 135, Gower street.

1883 MAUDSLEY, HENRY, M.D., Resident Medical Officer, University College Hospital, Gower street.

1839 MEADE, RICHARD HENRY, Consulting Surgeon to the Bradford Infirmary; Bradford, Yorkshire. *Trans.* 1.

1870 MEADOWS, ALFRED, M.D., Physician-Accoucheur to, and Lecturer on Midwifery and Diseases of Women and Children at, St. Mary's Hospital; 27, George street, Hanover square. *Lib. Com.* 1875-7.

1865 MEDWIN, AARON GEORGE, M.D., Consulting Dental Surgeon to the Royal Kent Dispensary, 34, Bruton street, Berkeley square, and 11, Montpellier row, Blackheath.

1880 MEREDITH, WILLIAM APPLETON, M.B., C.M., Surgeon to the Samaritan Free Hospital for Women and Children; 6, Queen Anne street, Cavendish square.

1874 MERRIMAN, JOHN J., 45, Kensington square.

1815 MEYER, AUGUSTUS, M.D., St. Petersburg.

1840 MIDDLEMORE, RICHARD, Consulting Surgeon to the Birmingham Eye Hospital; The Limes, Bristol road, Edgbaston, Birmingham.

1854 MIDDLESHIP, EDWARD ARCHIBALD.

1885 MILLICAN, KENNETH WILLIAM, B.A., 58, Welbeck street.

1882 MILLS, JOSEPH, 15, Henrietta street, Cavendish square.

1873 MILNER, EDWARD, Surgeon to the Lock Hospital; 32, New Cavendish street, Portland place.

1883 MONEY, ANGEL, M.D., Assistant Physician to the Hospital for Sick Children, Great Ormond Street, and to the City of London Hospital for Diseases of the Chest, Victoria park; 24, Harley street. *Trans.* 4.

1873 MOORE, NORMAN, M.D., Assistant Physician and Warden of the College, and Demonstrator of Morbid Anatomy, St. Bartholomew's Hospital; the College, St. Bartholomew's Hospital. *Referee*, 1886.

Elected

1857 MORGAN, JOHN, 3, Sussex place, Hyde park gardens. C. 1880-1. *Lib. Com.* 1862-3. *Trans.* 1.

1861 MORGAN, JOHN EDWARD, M.D., Physician to the Manchester Royal Infirmary, and Professor of Medicine in the Victoria University, Manchester; 1, St. Peter's square, Manchester.

1878 MORGAN, JOHN HAMMOND, M.A., Assistant Surgeon to the Charing Cross Hospital, and to the Hospital for Sick Children, Great Ormond street; 68, Grosvenor street. *Trans.* 1.

1874 MORRIS, HENRY, M.A., Surgeon to, and Lecturer on Surgery at, the Middlesex Hospital; 2, Mansfield street, Portland place. *Referee*, 1882-6. *Trans.* 9.

1879 MORRIS, MALCOLM ALEXANDER, Surgeon to the Skin Department of, and Lecturer on Dermatology at, St. Mary's Hospital; 63, Montagu square.

1885 MOTT, FREDERICK WALKER, M.B., Lecturer on Physiology, Charing Cross Hospital; Meadowlead, Gayton Road, Harrow.

1879 MUNK, WILLIAM, M.D., Harveian Librarian, Royal College of Physicians; Consulting Physician to the Royal Hospital for Incurables; 40, Finsbury square.

1873 MURRAY, J. IVOR, M.D., F.R.S.Ed. 24, Huntriss Row, Scarborough.

1880 MURRELL, WILLIAM, M.D., Assistant Physician to the Royal Hospital for Diseases of the Chest; Assistant Physician to, and Lecturer on Materia Medica and Therapeutics at, the Westminster Hospital; 38, Weymouth street, Portland place. *Trans.* 1.

1863 MYERS, ARTHUR BOWEN RICHARDS, Surgeon to the 1st Battalion, Coldstream Guards; 3, Park Terrace, Windsor. C. 1878-9. *Lib. Com.* 1877.

1882 MYERS, ARTHUR THOMAS, M.D., Medical Registrar, St. George's Hospital; 9, Lower Berkeley street, Portman square.

Elected

1881 NALL, SAMUEL, M.B., Disley, Stockport, Cheshire.

1870 NEILD, JAMES EDWARD, M.D., Lecturer on Forensic Medicine in the University of Melbourne; 166, Collins street east, Melbourne, Victoria.

1835 †NELSON, THOMAS ANDREW, M.D., 10, Nottingham terrace, York gate, Regent's park. *Lib. Com.* 1841.

1877 NETTLESHIP, EDWARD, Ophthalmic Surgeon to, and Lecturer on Ophthalmology at, St. Thomas's Hospital; Assistant Surgeon to the Royal London Ophthalmic Hospital; Ophthalmic Surgeon to the Hospital for Sick Children; 5, Wimpole street, Cavendish square.

1843 †NEWTON, EDWARD, 85, Gloucester terrace, Hyde Park. C. 1863-4.

1868 NICHOLLS, JAMES, M.D., Senior Medical Officer, Essex and Chelmsford Infirmary and Dispensary; the Old Infirmary, Chelmsford, Essex.

1849 NORMAN, HENRY BURFORD, Portland Lodge, Southsea, Hants. *Lib. Com.* 1857.

1847 *NOURSE, WILLIAM EDWARD CHARLES, Bouverie House, Exeter.

1864 NUNN, THOMAS WILLIAM, Consulting Surgeon to the Middlesex Hospital; 8, Stratford place, Oxford street.

1870 NUNNELEY, FREDERICK BARHAM, M.D. *Trans.* 2.

1884 OAKES, ARTHUR, M.D., 99, Priory road, West Hampstead.

1880 O'CONNOR, BERNARD, A.B., M.D., Physician to the North London Hospital for Consumption; 17, St. James's place.

1847 O'CONNOR, THOMAS, March, Cambridgeshire.

1880 OGILVIE, GEORGE, M.B., Lecturer on Experimental Physics at the Westminster Hospital; Physician to the Hospital for Epilepsy and Paralysis, Regent's Park; 13, Welbeck street, Cavendish square.

1880 OGILVIE, LESLIE, M.B., Lecturer on Comparative Anatomy at the Westminster Hospital; 46, Welbeck street, Cavendish square.

Elected

1858 OGLE, JOHN WILLIAM, M.D., *Vice-President*, Consulting Physician to St. George's Hospital; 30, Cavendish square. C. 1873. V.P. 1886. *Referee*, 1864-72. *Trans.* 4.

1855 *OGLE, WILLIAM, M.A., M.D., Physician to the Derbyshire Infirmary; The Elms, Duffield road, Derby.

1860 OGLE, WILLIAM, M.D., Superintendent of Statistics in the Registrar-General's Department, Somerset House; 10, Gordon street, Gordon square. S. 1868-70. C. 1876-7. *Lib. Com.* 1871-5. *Trans.* 5.

1870 OLDHAM, CHARLES FREDERIC, India [Agents: Messrs. Grindlay and Co., 55, Parliament street].

1883 *OLIVER, THOMAS, M.D., Lecturer on Practical Physiology, University of Durham; and Physician to the Newcastle-upon-Tyne Infirmary; 12, Eldon square, Newcastle-upon-Tyne.

1871 *O'NEILL, WILLIAM, M.D., Physician to the Lincoln Lunatic Hospital, Silver street, Lincoln.

1873 ORD, WILLIAM MILLER, M.D., Physician to, and Lecturer on Medicine at, St. Thomas's Hospital; 7, Upper Brook street, Grosvenor square. *Referee*, 1884-6. *Trans.* 6.

1877 ORMEROD, JOSEPH ARDERNE, M.D., Assistant Physician to the National Hospital for the Paralysed and Epileptic, Queen square, and to the City of London Hospital for Diseases of the Chest, Victoria Park; 25, Upper Wimpole street. *Trans.* 1.

1885 ORMSBY, L. HEPENSTAL, M.D., Lecturer on Clinical and Operative Surgery and Surgeon to the Meath Hospital and County Dublin Infirmary; Surgeon to the Children's Hospital, Dublin; 92, Merrion square west, Dublin.

1875 OSBORN, SAMUEL, 10, Maddox street, Regent street, and Maisonnette, Datchet, Bucks.

1879 OWEN, EDMUND, Surgeon to St. Mary's Hospital; Surgeon to the Hospital for Sick Children; 49, Seymour street, Portman square. *Trans.* 1.

Elected

1882 OWEN, HERBERT ISAMBARD, M.D., Assistant Physician to, and Lecturer on Materia Medica and Therapeutics at, St. George's Hospital; 5, Hertford street, May Fair.

1874 PAGE, HERBERT WILLIAM, M.A., M.C., Surgeon to, and Joint Lecturer on Surgery at, St. Mary's Hospital; 146, Harley street, Cavendish square. *Referee*, 1884-6. *Lib. Com.* 1886. *Trans.* 2.

1840 †PAGET, SIR JAMES, Bart., D.C.L., LL.D., F.R.S., Sergeant-Surgeon to H.M. the Queen; Surgeon-in-Ordinary to H.R.H. the Prince of Wales; Consulting Surgeon to St. Bartholomew's Hospital; Vice-Chancellor of the University of London; Foreign Associate of the 'Académie de Médecine,' Paris; 1, Harewood place, Hanover square. C. 1848-9. V.P. 1861. T. 1867. P. 1875-6. *Referee*, 1844-6, 1848, 1851-60, 1862-6, 1868-74. *Sci. Com.* 1863. *Lib. Com.* 1846-7. *Trans.* 12.

1858 *PALEY, WILLIAM, M.D., Physician to the Ripon Dispensary; The Old Residence, Ripon, Yorkshire.

1847 PARKER, NICHOLAS, M.D., Paris.

1873 PARKER, ROBERT WILLIAM, Surgeon to the East London Hospital for Children; 8, Old Cavendish street. *Lib. Com.* 1885-6. *Trans.* 3.

1885 PARKER, RUSHTON, M.B., Professor of Surgery, University College, Liverpool (Victoria University); Assistant Surgeon to the Liverpool Royal Infirmary; 59, Rodney street, Liverpool.

1883 PASTEUR, WILLIAM, M.D., Medical Registrar to the Middlesex Hospital; Physician to the North-Eastern Hospital for Children; 19, Queen street, May Fair.

1865 PAVY, FREDERICK WILLIAM, M.D., F.R.S., Physician to Guy's Hospital; 35, Grosvenor street. C. 1883-4. *Referee*, 1871-82. *Trans.* 1.

1869 PAYNE, JOSEPH FRANK, M.D., Senior Assistant-Physician to, and Lecturer on Pathological Anatomy at, St. Thomas's Hospital; 78, Wimpole street, Cavendish square. *Sci. Com.* 1879. *Lib. Com.* 1878-85.

Elected

1879 PEEL, ROBERT, 120, Collins street east, Melbourne, Victoria.

1856 PEIRCE, RICHARD KING, Woodside, Windsor forest, Berks.

1830 PELECHIN, CHARLES P., M.D., St. Petersburg.

1855 *PEMBERTON, OLIVER, Senior Surgeon to the Birmingham General Hospital, and Professor of Surgery at the Queen's College, Birmingham; 12, Temple row, Birmingham. *Trans.* 1.

1874 PENHALL, JOHN THOMAS, 5, Eversfield place, St. Leonard's Sussex.

1870 PERRIN, JOHN BESWICK, Vernon House, Leigh, Lancashire.

1879 *PESIKAKA, HORMASJI DOSABHAI, Marine Lines, Bombay.

1878 *PHILIPSON, GEORGE HARE, M.D., M.A., D.C.L., Professor of Medicine at Durham University; Senior Physician to the Newcastle-upon-Tyne Infirmary; 7, Eldon square, Newcastle-upon-Tyne.

1883 PHILLIPS, CHARLES DOUGLAS F., M.D., F.R.S.Ed., 10, Henrietta street, Cavendish square, W.

1884 PHILLIPS, GEORGE RICHARD TURNER, 24, Leinster square, Bayswater.

1867 PICK, THOMAS PICKERING, Surgeon to, and Lecturer on Surgery at, St. George's Hospital; 18, Portman street, Portman square. C. 1884-5. *Referee,* 1882-3. *Sci. Com.* 1870. *Lib. Com.* 1879-81.

1841 †PITMAN, SIR HENRY ALFRED, M.D., Consulting Physician to St. George's Hospital; 28, Gordon square. L. 1851-3. C. 1861-2. T. 1863-8. V.P. 1870-1. *Referee,* 1849-50. *Lib. Com.* 1847.

1884 PITT, GEORGE NEWTON, M.D., Medical Registrar and Demonstrator of Practical Medicine at Guy's Hospital; 34, Ashburn place, South Kensington.

1885 POLAND, JOHN, Demonstrator of Anatomy, Guy's Hospital; 16, St. Thomas's street, Southwark.

1884 POLLARD, BILTON, M.D., Surgical Registrar, University College Hospital; 50, Torrington square.

Elected

1871 POLLOCK, ARTHUR JULIUS, M.D., Senior Physician to, and Lecturer on the Principles and Practice of Medicine at, Charing Cross Hospital; Physician to the Foundling Hospital; 85, Harley street, Cavendish square.

1845 †POLLOCK, GEORGE DAVID, *President*, Surgeon-in-Ordinary to H.R.H. the Prince of Wales; Consulting Surgeon to St. George's Hospital; 36, Grosvenor street. C. 1856-7. L. 1859-62. V.P. 1870-1. P. 1886. *Referee*, 1858, 1864-9, 1877-85. *Trans.* 5.

1865 POLLOCK, JAMES EDWARD, M.D., Consulting Physician to the Hospital for Consumption, Brompton; 52, Upper Brook street, Grosvenor square. C. 1882-3. *Referee*, 1872-81.

1871 POORE, GEORGE VIVIAN, M.D., Professor of Medical Jurisprudence in University College, London; Physician to University College Hospital; Consulting Physician to the Royal Infirmary for Children and Women, Waterloo road; 30, Wimpole street. *Trans.* 1.

1885 PORT, HEINRICH, M.D., Physician to the German Hospital; 48, Finsbury square.

1846 POTTER, JEPHSON, M.D., F.L.S.

1842 POWELL, JAMES, M.D.

1867 POWELL, RICHARD DOUGLAS, M.D., Physician to, and Lecturer on Practical Medicine at, the Middlesex Hospital; Physician to the Hospital for Consumption and Diseases of the Chest, Brompton; 62, Wimpole st., Cavendish sq. S. (Oct.) 1883-5. *Referee*, 1879-83, 1886. *Trans.* 2.

1867 POWER, HENRY, Senior Ophthalmic Surgeon to, and Lecturer on Ophthalmic Surgery at, St. Bartholomew's Hospital; 37A, Great Cumberland place, Hyde park. C. 1882-3. *Referee*, 1870-81. *Sci. Com.* 1870. *Lib. Com.* 1872-8.

1857 PRIESTLEY, WILLIAM OVEREND, M.D., LL.D., Consulting Physician to King's College Hospital, and to the St. Marylebone Infirmary; 17, Hertford street, Mayfair. C. 1874-5. V.P. 1884-5. *Referee*, 1867-73, 1877-83. *Sci. Com.* 1863.

Elected

1883 PRINGLE, JOHN JAMES, M.B., C.M., Assistant Physician to the Middlesex Hospital, and Physician to the Royal Hospital for Diseases of the Chest; 35, Bruton street, Berkeley square.

1874 PURVES, WILLIAM LAIDLAW, Aural Surgeon to Guy's Hospital; 20, Stratford place, Oxford street. *Trans.* 2.

1878 PYE, WALTER, Surgeon (with charge of out-patients) to St. Mary's Hospital and to the Victoria Hospital for Children; 4, Sackville street, Piccadilly.

1877 PYE-SMITH, PHILIP HENRY, M.D., F.R.S., Physician to, and Lecturer on Medicine at, Guy's Hospital; Member of the Senate of the University of London; 54, Harley street, Cavendish square.

1850 †QUAIN, RICHARD, M.D., F.R.S., Consulting Physician to the Hospital for Consumption, Brompton; Member of the Senate of the University of London; 67, Harley street, Cavendish square. C. 1866-7. V.P. 1878-9. *Sci. Com.* 1863. *Trans.* 1.

1835 †QUAIN, RICHARD, F.R.S., Surgeon-Extraordinary to H.M. the Queen; Emeritus Professor of Clinical Surgery, University College, London, and Consulting Surgeon to University College Hospital; 32, Cavendish square. C. 1838-9. L. 1846-8. T. 1851-3. V.P. 1856-7. *Referee*, 1845-6, 1848, 1858-9. *Lib. Com.* 1846. *Trans.* 1. *Pro.* 2.

1852 †RADCLIFFE, CHARLES BLAND, M.D., *Treasurer*, Consulting Physician to the Westminster Hospital; Physician to the National Hospital for the Paralysed and Epileptic; 25, Cavendish square. C. 1867-8. V.P. 1879-80. T. 1881-6. *Referee*, 1862-6, 1870-8.

1871 RALFE, CHARLES HENRY, M.D., M.A., Assistant Physician to the London Hospital, and late Physician to the Seamen's Hospital, Greenwich; 26, Queen Anne street, Cavendish square. *Referee*, 1885-6.

1857 RANKE, HENRY, M.D., 3, Sophienstrasse, Munich.

1854 RANSOM, WILLIAM HENRY, M.D., F.R.S., Physician to the Nottingham General Hospital, Nottingham.

Elected

1869 READ, THOMAS LAURENCE, 11, Petersham terrace, Queen's gate.

1858 REED, FREDERICK GEORGE, M.D., 46, Hertford street, Mayfair. *Trans.* 1.

1821 REEDER, HENRY, M.D., Varick, Seneca County, New York, United States.

1857 REES, GEORGE OWEN, M.D., F.R.S., Physician Extraordinary to H.M. the Queen, Consulting Physician to Guy's Hospital; 26, Albemarle street, Piccadilly. C. 1873. *Referee*, 1860-72, 1875-81. *Trans.* 1.

1882 REID, JAMES, M.D., Resident Physician to H.M. the Queen, Windsor Castle.

1884 REID, THOMAS WHITEHEAD, Surgeon to the Kent and Canterbury Hospital; 34, St. George's place, Canterbury.

1855 REYNOLDS, JOHN RUSSELL, M.D., F.R.S., *Vice-President*, Physician-in-Ordinary to H.M.'s Household; Consulting-Physician to University College Hospital; 38, Grosvenor street. C. 1870. V.P. 1883. *Referee*, 1867-9.

1865 RHODES, GEORGE WINTER, Surgeon to the Huddersfield Infirmary; Queen street south, Huddersfield.

1881 RICE, GEORGE, M.B., C.M., Sutton, Surrey.

1852 RICHARDSON, CHRISTOPHER THOMAS, M.B., 13, Nelson crescent, Ramsgate.

1845 †RIDGE, BENJAMIN, M.D., 8, Mount street, Grosvenor square.

1863 RINGER, SYDNEY, M.D., F.R.S., Professor of the Principles and Practice of Medicine in University College, London, and Physician to University College Hospital; 15, Cavendish place, Cavendish square. C. 1881-2. *Referee*, 1873-80. *Trans.* 6.

1871 RIVINGTON, WALTER, M.S., Surgeon to, and Lecturer on Surgery at, the London Hospital; 22, Finsbury square. C. 1885-6. *Trans.* 4.

Elected

1871 *ROBERTS, DAVID LLOYD, M.D., Obstetric Physician to the Manchester Royal Infirmary, Physician to St. Mary's Hospital, Manchester; 11, St. John street, Manchester.

1878 ROBERTS, FREDERICK THOMAS, M.D., Professor of Materia Medica and Therapeutics in University College, London; and Physician to University College Hospital; Physician to the Hospital for Consumption, Brompton; 102, Harley street, Cavendish square.

1857 ROBERTSON, JOHN CHARLES GEORGE, Medical Superintendent of the Cavan District Lunatic Asylum; Monaghan, Ireland.

1873 ROBERTSON, WILLIAM HENRY, M.D., Consulting Physician to the Buxton Bath Charity and Devonshire Hospital; Buxton, Derbyshire.

1885 ROCKWOOD, WILLIAM GABRIEL, M.D., Colombo, Ceylon.

1850 ROPER, GEORGE, M.D., Consulting Physician to the Eastern Division of the Royal Maternity Charity; Physician to the Royal Infirmary for Children and Women, Waterloo Bridge road; 19, Ovington gardens. C. 1879-80.

1857 ROSE, HENRY COOPER, M.D., F.L.S., Consulting Surgeon to the Hampstead Dispensary; Penrose House, Hampstead. C. 1886. *Trans.* 1.

1883 ROSE, WILLIAM, M.B., Surgeon to King's College Hospital and to the Royal Free Hospital; 50, Harley street, Cavendish square.

1882 ROUTH, AMAND JULES MCCONNEL, M.D., B.S., Physician to the Samaritan Free Hospital for Women; Assistant Obstetric Physician to the Charing Cross Hospital; Obstetric Physician to the St. Marylebone General Dispensary; 6, Upper Montagu street, Montagu square.

1849 †ROUTH, CHARLES HENRY FELIX, M.D., Consulting Physician to the Samaritan Free Hospital for Women and Children; 52, Montagu square. *Lib. Com.* 1854-5. *Trans.* 1.

Elected

1863 ROWE, THOMAS SMITH, M.D., Senior Visiting Surgeon to the Royal Sea-Bathing Infirmary; Cecil street, Margate, Kent.

1882 ROY, CHARLES SMART, M.D., F.R.S., Professor of Pathology in the University of Cambridge; Trinity College, Cambridge.

1871 RUTHERFORD, WILLIAM, M.D., F.R.S., Professor of the Institutes of Medicine in the University of Edinburgh; 14, Douglas crescent, Edinburgh.

1886 SAINSBURY, HARRINGTON, M.D., Assistant Physician and Pathologist to the Royal Free Hospital; 63, Welbeck street, Cavendish square. *Trans.* 1.

1856 SALTER, S. JAMES A., M.B., F.R.S., F.L.S., Basingfield, near Basingstoke, Hants. C. 1871. *Lib. Com.* 1878. *Trans.* 2.

1849 †SANDERSON, HUGH JAMES, M.D., 26, Upper Berkeley street, Portman square. C. 1872-3. *Lib. Com.* 1862-3.

1855 SANDERSON, JOHN BURDON, M.D., LL.D., F.R.S., Waynflete Professor of Physiology in the University of Oxford; 50, Banbury road, Oxford. C. 1869-70. V.P. 1882. *Referee*, 1867-8, 1876-81. *Sci. Com.* 1862, 1870. *Lib. Com.* 1876-81. *Trans.* 2.

1867 SANDFORD, FOLLIOTT JAMES, M.D., Market Drayton, Shropshire.

1879 SANGSTER, ALFRED, B.A., M.B., Physician to the Skin Department, and Demonstrator of Skin Diseases at the Charing Cross Hospital; 6, Savile row. *Trans.* 1.

1847 †SANKEY, WILLIAM HENRY OCTAVIUS, M.D., Boreatton park, Baschurch, near Shrewsbury.

1869 SANSOM, ARTHUR ERNEST, M.D., Senior Physician to the North-Eastern Hospital for Children; Physician (with charge of out-patients) to the London Hospital; 84, Harley street, Cavendish square. *Trans.* 2.

1845 †SAUNDERS, SIR EDWIN, Surgeon-Dentist to H.M. the Queen, and to their R.H. the Prince and Princess of Wales; 13A, George street, Hanover square. C. 1872-3.

Elected

1834 SAUVAN, LUDWIG V., M.D., Warsaw.

1879 SAVAGE, GEORGE HENRY, M.D., Medical Superintendent and Resident Physician to the Bethlem Royal Hospital, St. George's road, Southwark.

1859 SAVORY, WILLIAM SCOVELL, F.R.S., Surgeon to, and Lecturer on Surgery at, St. Bartholomew's Hospital; Surgeon to Christ's Hospital; 66, Brook street, Grosvenor square. C. 1871-2. L. 1878. V.P. 1883-4. *Referee*, 1865-70, 1873-77, 1879-82. *Sci. Com.* 1862, 1867, 1870. *Lib. Com.* 1866-8. *Trans.* 7.

1883 SCHÄFER, EDWARD ALBERT, F.R.S., Jodrell Professor of Physiology, University College, London; University College, Gower street.

1861 *SCOTT, WILLIAM, M.D., Senior Physician to the Huddersfield Infirmary; Waverley House, Huddersfield.

1882 SCRIVEN, JOHN BARCLAY, Brigade Surgeon, Bengal (retired), late Professor of Anatomy, Surgery, and Ophthalmic Surgery at the Lahore Medical School; 95, Oxford gardens, Notting hill.

1863 SEDGWICK, WILLIAM, 12, Park place, Upper Baker street. C. 1884-5. *Trans.* 3.

1877 SEMON, FELIX, M.D., Assistant Physician for Diseases of the Throat to St. Thomas's Hospital; 39, Wimpole street, Cavendish square. *Trans.* 1.

1875 SEMPLE, ROBERT HUNTER, M.D., Physician to the Bloomsbury Dispensary; 8, Torrington square. *Sci. Com.* 1879.

1873 *SHAPTER, LEWIS, B.A., M.B., Physician to the Devon and Exeter Hospital; the Barnfield, Exeter.

1882 SHARKEY, SEYMOUR JOHN, M.B., Assistant Physician, Joint Lecturer on Pathology, and Demonstrator of Morbid Anatomy, to St. Thomas's Hospital; 2, Portland place. *Trans.* 2.

1840 SHARP, WILLIAM, M.D., F.R.S., Horton House, Rugby. *Trans.* 1.

Elected

1836 †SHAW, ALEXANDER, Consulting Surgeon to the Middlesex Hospital; 136, Abbey road, Kilburn. C. 1842. S. 1843-4. V.P. 1851-2. T. 1858-60. *Referee*, 1842-3, 1846-50, 1855-7, 1865. *Lib. Com.* 1843. *Trans.* 4.

1886 SHAW, LAURESTON ELGIE, M.D., 3, Newton grove, Bedford park.

1884 SHEILD, ARTHUR MARMADUKE, M.B., B.S., House Surgeon, St. George's Hospital.

1859 SIBLEY, SEPTIMUS WILLIAM, 7, Harley street, Cavendish square. C. 1882-3. *Sci. Com.* 1863. *Trans.* 4.

1848 †SIEVEKING, SIR EDWARD HENRY, M.D., Physician-Extraordinary to H.M. the Queen; Physician-in-Ordinary to H.R.H. the Prince of Wales; Physician to St. Mary's Hospital; 17, Manchester square. C. 1859-60. S. 1861-3. V.P. 1873-4. L. 1881-2. *Referee*, 1855-8, 1864-72, 1875-80. *Sci. Com.* 1862. *Trans.* 2.

1842 †SIMON, JOHN, C.B., D.C.L., LL.D., F.R.S., Consulting Surgeon to St. Thomas's Hospital; 40, Kensington square. C. 1854-5. V.P. 1865. *Referee* 1851-3, 1866-81. *Trans.* 1.

1857 SIORDET, JAMES LEWIS, M.B., Villa Preti, Mentone, Alpes Maritimes, France.

1882 SMITH, CHARLES JOHN, 54, Old Steyne, Brighton.

1879 SMITH, E. NOBLE, Senior Surgeon and Surgeon to the Orthopædic Department of the Farringdon Dispensary; Orthopædic Surgeon to the British Home for Incurables; 24, Queen Anne street, Cavendish square.

1881 SMITH, EUSTACE, M.D., Physician to H.M. the King of the Belgians; Physician to the East London Children's Hospital, and to the Victoria Park Hospital for Diseases of the Chest; 5, George street, Hanover square.

1885 SMITH, JAMES GREIG, M.B., C.M., F.R.S.Ed., Surgeon to the Bristol Royal Infirmary; 16, Victoria square, Clifton.

Elected

1872 SMITH, T. GILBART, M.A., M.D., Assistant-Physician to the London Hospital; Physician to the Royal Hospital for Diseases of the Chest, City road; 68, Harley street, Cavendish square. *Trans.* 1.

1866 SMITH, HEYWOOD, M.A. M.D., Physician to the Hospital for Women; Physician to the British Lying-in Hospital; 18, Harley street, Cavendish square.

1838 †SMITH, SPENCER, Consulting Surgeon to St. Mary's Hospital; 92, Oxford terrace, Hyde Park. C. 1854. S. 1855-8. V.P. 1859-60. T. 1865. *Referee*, 1851-3, 1862-4, 1866-78. *Lib. Com.* 1847.

1863 SMITH, THOMAS, Surgeon to, and Lecturer on Clinical Surgery at, St. Bartholomew's Hospital; 5, Stratford place, Oxford street. S. 1870-2. C. 1875-6. *Referee*, 1873-4, 1880-6. *Sci. Com.* 1867. *Trans.* 3.

1873 SMITH, W. JOHNSON, Surgeon to the Seamen's Hospital, Greenwich.

1874 *SMITH, WILLIAM ROBERT, M.D., D.Sc., F.R.S.Ed., 74, Great Russell Street, Bloomsbury.

1868 SOLLY, SAMUEL EDWIN, Colorado Springs, Colorado, U.S.

1865 SOUTHEY, REGINALD, M.D., Commissioner in Lunacy; 32, Grosvenor road, Westminster. C. 1881-2. S. 1883. *Referee*, 1873-80. *Trans.* 1.

1844 SPACKMAN, FREDERICK ROBERT, M.D., Consulting Physician to St. Alban's Hospital, Harpenden, St. Alban's.

1875 SPITTA, EDMUND JOHNSON, Ivy House, Clapham Common, Surrey.

1851 †SPITTA, ROBERT JOHN, M.D., East Side, Clapham Common, Surrey. C. 1878-9. *Trans.* 1.

1885 SQUIRE, JOHN EDWARD, M.D., Assistant Physician to the North London Hospital for Consumption; 23, Seymour street, Portman square. *Trans.* 1.

1882 STEAVENSON, WILLIAM EDWARD, M.D., Electrician to St. Bartholomew's Hospital; Physician to the Alexandra Hospital for Children; 39, Welbeck street, Cavendish square.

Elected

1854 STEVENS, HENRY, M.D., Inspector, Medical Department, Local Government Board, Whitehall.

1884 STEWART, EDWARD, M.D., 16, Harley street.

1859 STEWART, WILLIAM EDWARD, 16, Harley street, Cavendish square.

1879 *STIRLING, EDWARD CHARLES, late Assistant Surgeon and Lecturer on Physiology at St. George's Hospital; Adelaide, South Australia [care of T. Gemmell, Esq., 11, Essex street, Strand].

1856 STOCKER, ALONZO HENRY, M.D., Peckham House, Peckham.

1865 STOKES, SIR WILLIAM, M.D., M.C., Surgeon to the Richmond Surgical Hospital; 5, Merrion square north, Dublin. *Trans.* 1.

1884 STONHAM, CHARLES, Curator of the Pathological Museum, University College, London, and Assistant Surgeon to the Cancer Hospital, Brompton; 109, Gower street.

1843 STORKS, ROBERT REEVE, Paris.

1871 STRONG, HENRY JOHN, M.D., Surgeon to the Croydon General Hospital; Whitgift House, George street, Croydon.

1863 †STURGES, OCTAVIUS, M.D., Physician to, and Lecturer on Medicine at, the Westminster Hospital; Physician to the Hospital for Sick Children; 85, Wimpole street, Cavendish square. C. 1878-9. *Referee*, 1882-6.

1871 †SUTHERLAND, HENRY, M.D., Lecturer on Insanity at the Westminster Hospital; 6, Richmond terrace, Whitehall.

1871 SUTTON, HENRY GAWEN, M.B., Physician to, and Lecturer on Pathology at, the London Hospital, and Physician to the London Hospital; 9, Finsbury square. *Trans.* 1.

1883 SUTTON, JOHN BLAND, Assistant Surgeon, Lecturer on Comparative Anatomy, and Senior Demonstrator of Anatomy to the Middlesex Hospital; 22, Gordon street, Gordon square. *Trans.* 3.

1861 *SWEETING, GEORGE BACON, Consulting Surgeon to the West Norfolk Hospital; King's Lynn, Norfolk.

Elected

1886 SYMONDS, CHARTERS JAMES, M.S., Assistant Surgeon to Guy's Hospital; 26, Weymouth street, Portland place.

1878 *SYMPSON, THOMAS, Surgeon to the Lincoln County Hospital; 3, James street, Lincoln.

1870 TAIT, LAWSON, Surgeon to the Birmingham and Midland Hospital for Women; 7, The Crescent, Birmingham. *Trans.* 4.

1864 TAUSSIG, GABRIEL, M.D., 70, Piazza Barberini, Rome.

1875 TAY, WAREN, Surgeon to the London Hospital, to the Royal London Ophthalmic Hospital, to the North Eastern Hospital for Children, and to the Hospital for Skin Diseases, Blackfriars; 4, Finsbury square.

1873 TAYLOR, FREDERICK, M.D., Physician to, and Lecturer on Materia Medica at, Guy's Hospital; Physician to the Evelina Hospital for Sick Children; 11, St. Thomas's street, Southwark. *Trans.* 1.

1845 †TAYLOR, THOMAS, Warwick House, 1, Warwick place, Grove End road, St. John's wood.

1859 TEGART, EDWARD, 49, Jermyn street, St. James's.

1874 THIN, GEORGE, M.D., 22, Queen Anne street, Cavendish square. *Trans.* 9.

1862 THOMPSON, EDMUND SYMES, M.D., Senior Physician to the Hospital for Consumption, Brompton; Gresham Professor of Medicine; 33, Cavendish square. S. 1871-4. C. 1878-9. *Referee*, 1876-7. *Trans.* 1.

1857 THOMPSON, HENRY, M.D., Consulting Physician to the Middlesex Hospital; 53, Queen Anne street, Cavendish square.

1852 †THOMPSON, SIR HENRY, Surgeon-Extraordinary to H.M. the King of the Belgians; Emeritus Professor of Clinical Surgery in University College, London; and Consulting Surgeon to University College Hospital; Corresponding Member of the "Société de Chirurgie," Paris; 35, Wimpole street, Cavendish square. C. 1869. *Trans.* 7.

Elected

1862 THOMPSON, REGINALD EDWARD, M.D., Physician to the Hospital for Consumption, Brompton; 47, Park street, Grosvenor square. C. 1879. S. 1880-82. V.P. 1883-4. *Referee*, 1873-8. *Sci. Com.* 1867. *Trans.* 2.

1881 THOMSON, WILLIAM SINCLAIR, M.D., 40, Ladbroke grove, Kensington park gardens.

1876 THORNTON, JOHN KNOWSLEY, M.B., C.M., Surgeon to the Samaritan Free Hospital for Women and Children; 22, Portman street, Portman square. *Lib. Com.* 1886. *Trans.* 3.

1883 THURSFIELD, THOMAS WILLIAM, M.D., Physician to the Warneford and South Warwickshire General Hospital; 26, The Parade, Leamington.

1848 †TILT, EDWARD JOHN, M.D., Consulting Physician to the Farringdon General Dispensary and Lying-in Charity; 27, Seymour street, Portman square. *Referee*, 1874-81.

1880 TIVY, WILLIAM JAMES, 8, Lansdowne place, Clifton, Bristol.

1872 TOMES, CHARLES SISSMORE, M.A., F.R.S., 37, Cavendish square. *Lib. Com.* 1879.

1867 TONGE, MORRIS, M.D., Harrow-on-the-Hill, Middlesex.

1882 TOOTH, HOWARD HENRY, M.B., Assistant Demonstrator of Practical Physiology, St. Bartholomew's Hospital; 34, Harley street, Cavendish square.

1871 *TREND, THEOPHILUS W., M.D., Raeberry Lodge, Southampton.

1879 TREVES, FREDERICK, Surgeon to, and Lecturer on Anatomy at, the London Hospital; 6, Wimpole street, Cavendish square. *Trans.* 3.

1881 *TREVES, WILLIAM KNIGHT, Surgeon to the National Hospital for Scrofula; 31, Dalby square, Cliftonville, Margate.

1867 TROTTER, JOHN WILLIAM, late Surgeon-Major, Coldstream Guards; 4, St. Peter's terrace, York.

1859 TRUMAN, EDWIN THOMAS, Surgeon-Dentist in Ordinary to Her Majesty's Household; 23, Old Burlington street.

Elected

1862 TUKE, THOMAS HARRINGTON, M.D., Manor House, Chiswick, and 37, Albemarle street, Piccadilly.

1875 TURNER, FRANCIS CHARLEWOOD, M.A., M.D., Physician to the North-Eastern Hospital for Children, and to the London Hospital; 15, Finsbury square.

1873 TURNER, GEORGE BROWN, M.D., San Remo, Italy.

1882 TURNER, GEORGE ROBERTSON, Visiting Surgeon to the Seamen's Hospital, Greenwich; Demonstrator of Anatomy and Joint Lecturer on Practical Surgery at St. George's Hospital; 49, Green street, Park lane.

1881 TYSON, WILLIAM JOSEPH, M.D., Medical Officer of the Folkestone Infirmary; 10, Langhorne gardens, Folkestone.

1876 VENN, ALBERT JOHN, M.D., Obstetric Physician to the Metropolitan Free Hospital; Physician to the Victoria Hospital for Children, Chelsea; and Assistant Physician for the Diseases of Women, West London Hospital; 8, Upper Brook street, Grosvenor square.

1870 VENNING, EDGCOMBE, 30, Cadogan place.

1865 VERNON, BOWATER JOHN, Ophthalmic Surgeon to St. Bartholomew's Hospital and to the West London Hospital; 14, Clarges street, Piccadilly.

1867 VINTRAS, ACHILLE, M.D., Physician to the French Embassy, and to the French Hospital, Leicester place; 19A, Hanover square.

1828 VULPES, BENEDETTO, M.D., Physician to the Hospital of Aversa, and the Hospital of Incurables, Naples.

1854 WADDINGTON, EDWARD, Hamilton, Auckland, New Zealand.

1870 WADHAM, WILLIAM, M.D., Physician to St. George's Hospital; 14, Park lane.

1886 WAINEWRIGHT, BENJAMIN, M.B., C.M., 6, Harley street, Cavendish square.

1864 WAITE, CHARLES DERBY, M.B., Consulting Physician to the Westminster General Dispensary; 3, Old Burlington street.

Elected.

1884 WAKLEY, Thomas, jun., 96, Redcliffe gardens.

1868 *WALKER, ROBERT, Honorary Surgeon to the Carlisle Dispensary; 2, Portland square, Carlisle.

1883 WALLER, AUGUSTUS, M.D., Lecturer on Physiology, St. Mary's Hospital; 29, Abbey road, St. John's wood.

1867 *WALLIS, GEORGE, Surgeon to Addenbrooke's Hospital, Corpus Buildings, Cambridge.

1873 WALSHAM, WILLIAM JOHNSON, C.M., Assistant Surgeon to, and Demonstrator of Practical and Orthopædic Surgery at, St. Bartholomew's Hospital; Surgeon to the Metropolitan Free Hospital; 27, Weymouth street, Portland place. *Lib. Com.* 1882-5. *Trans.* 3.

1852 †WALSHE, WALTER HAYLE, M.D., Emeritus Professor of the Principles and Practice of Medicine, University College, London; Consulting Physician to the Hospital for Consumption and to University College Hospital; 41, Hyde park square. C. 1872. *Trans.* 1.

1883 *WALTERS, JAMES HOPKINS, 15, Friar street, Reading.

1851 †WALTON, HAYNES, Consulting Surgeon to St. Mary's Hospital, 1, Brook street, Grosvenor square. *Trans.* 1. *Pro.* 1.

1852 WANE, DANIEL, M.D.

1821 WARD, WILLIAM TILLEARD, Tilleards, Stanhope, Canada.

1846 WARE, JAMES THOMAS, Tilford House, near Farnham, Surrey.

1866 WARING, EDWARD JOHN, C.I.E., M.D., 49, Clifton gardens, Maida vale. *Referee*, 1881-5.

1877 WARNER, FRANCIS, M.D., Assistant Physician and Lecturer on Botany to the London Hospital; 24, Harley street, Cavendish square. *Trans.* 1.

1861 WATERS, A. T. HOUGHTON, M.D., Physician to the Royal Infirmary; 69, Bedford street, Liverpool. *Trans.* 3.

1879 WATERS, JOHN HENRY, M.D., C.M., 101, Jermyn street.

1878 WATNEY, HERBERT, M.D., 1, Wilton crescent, Belgrave square, and Buckhold, Basildon, Reading.

Elected

1861 †WATSON, WILLIAM SPENCER, M.B., Surgeon to the Great Northern Hospital; Surgeon to the Royal South London Ophthalmic Hospital; 7, Henrietta street, Cavendish square. C. 1883-4. *Trans.* 1.

1879 DE WATTEVILLE, ARMAND, M.A., M.D., B.Sc., Medical Electrician to St. Mary's Hospital; 30, Welbeck street, Cavendish square.

1854 WEBB, WILLIAM, M.D., Gilkin View House, Wirksworth, Derbyshire.

1840 WEBB, WILLIAM WOODHAM, M.D., 82, Avenue des Termes, Paris.

1857 WEBER, HERMANN, M.D., *Vice-President*, Physician to the German Hospital; 10, Grosvenor street, Grosvenor square. C. 1874-5. V.P. 1885-6. *Referee*, 1869-73, 1878-84. *Lib. Com.* 1864-73. *Trans.* 6.

1844 †WEGG, WILLIAM, M.D., 15, Hertford street, Mayfair. L. 1854-8. C. 1861-2. T. 1873-80. *Lib. Com.* 1851-3.

1878 WEISS, HUBERT FOVEAUX, Assistant Surgeon to the West London Hospital; 11, Hanover square.

1874 WELLS, HARRY, M.D., San Ysidro, Buenos Ayres, S. America.

1854 †WELLS, SIR THOMAS SPENCER, Bart., Surgeon-in-Ordinary to H.M.'s Household; Consulting Surgeon to the Samaritan Free Hospital for Women and Children; 3, Upper Grosvenor street. C. 1870. V.P. 1881. *Trans.* 13. *Pro.* 1.

1842 †WEST, CHARLES, M.D., Corresponding Member of the Academy of Medicine of Paris; 55, Harley street, Cavendish square. C. 1855-6. V.P. 1863. P. 1877-8. *Referee*, 1848-54, 1857-62, 1864-76, 1880. *Sci. Com.* 1863. *Lib. Com.* 1844-7, 1851. *Trans.* 2.

1877 WEST, SAMUEL, M.D., Physician and Pathologist to the City of London Hospital for Diseases of the Chest, Victoria Park; Physician to the Royal Free Hospital; Medical Registrar and Medical Tutor to St. Bartholomew's Hospital; 15, Wimpole street, Cavendish square. *Trans.* 3.

Elected

1882 WHARRY, CHARLES JOHN, M.D., Resident Superintendent, Government Civil Hospital, Hong Kong.

1881 WHARRY, ROBERT, M.D., Physician to the Westminster Dispensary; 6, Gordon square.

1878 WHARTON, HENRY THORNTON, M.A., Honorary Surgeon to the Kilburn Dispensary; 39, St. George's road, Kilburn.

1828 WHATLEY, JOHN, M.D.

1875 WHIPHAM, THOMAS TILLYER, M.B., Physician to, and Lecturer on Pathology and Practical Medicine at, St. George's Hospital; 11, Grosvenor street, Grosvenor square.

1849 WHITE, JOHN.

1881 WHITE, WILLIAM HALE, M.D., Assistant Physician to Guy's Hospital; 4, St. Thomas's street, Southwark. *Trans.* 1.

1881 *WHITEHEAD, WALTER, F.R.S. Ed., Surgeon to the Manchester Royal Infirmary; Senior Surgeon to the Manchester and Salford Lock and Skin Hospital; 24, St. Ann's square, Manchester. *Trans.* 1.

1885 *WHITLA, WILLIAM, M.D., Physician to, and Lecturer in Medicine at, the Belfast Royal Hospital; Consulting Physician to the Ulster Hospital for Women and Children; 8, College square north, Belfast.

1877 WHITMORE, WILLIAM TICKLE, Surgeon to the Westminster General Dispensary; 7, Arlington street, Piccadilly.

1852 WIBLIN, JOHN, M.D., Medical Inspector of Emigrants and Recruits; Southampton. *Trans.* 1.

1870 *WILKIN, JOHN F., M.D., M.C., The Warren, Beckenham park, Kent.

1883 *WILKINSON, THOMAS MARSHALL, Surgeon to the Lincoln County Hospital and to the Lincoln General Dispensary; 7, Lindum road, Lincoln.

1837 WILKS, GEORGE AUGUSTUS FREDERICK, M.D., Stanbury, Torquay.

Elected

1863 WILKS, SAMUEL, M.D., LL.D., F.R.S., Physician in Ordinary to their Royal Highnesses the Duke and Duchess of Connaught, and to H.R.H. the Duke of Edinburgh; Consulting Physician to Guy's Hospital, and Member of the Senate of the University of London; 72, Grosvenor street, Grosvenor square. *Referee*, 1872-81. *Sci. Com.* 1.

1883 *WILLANS, WILLIAM BLUNDELL, Great Hadham, Herts.

1865 †WILLETT, ALFRED, Surgeon to St. Bartholomew's Hospital; Surgeon to St. Luke's Hospital; 36, Wimpole street, Cavendish square. C. 1880-81. *Referee*, 1882-6. *Trans.* 2.

1864 WILLETT, EDMUND SPARSHALL, M.D., Resident Physician, Wyke House, Isleworth, Middlesex.

1840 †WILLIAMS, CHARLES JAMES BLASIUS, M.D., F.R.S., Physician-Extraordinary to H.M. the Queen; Consulting Physician to the Hospital for Consumption, Brompton [47, Upper Brook street, Grosvenor square]; Villa de Rocher, Cannes. C. 1849-50. V.P. 1860-1. P. 1873-4. *Referee*, 1843-4. *Sci. Com.* 1862. *Trans.* 1.

1859 *WILLIAMS, CHARLES, Surgeon to the Norfolk and Norwich Hospital; 48, Prince of Wales road, Norwich.

1866 WILLIAMS, CHARLES THEODORE, M.A., M.D., Physician to the Hospital for Consumption and Diseases of the Chest, Brompton; 47, Upper Brook street, Grosvenor square. C. 1884-5. *Lib. Com.* 1880-3. *Trans.* 4.

1881 WILLIAMS, DAWSON, M.D., Assistant Physician to the East London Hospital for Children; 4, Oxford and Cambridge Mansions, Marylebone road.

1872 WILLIAMS, JOHN, M.D., Obstetric Physician to University College Hospital; Examiner in Obstetric Medicine at the University of London; 11, Queen Anne street, Cavendish square. *Referee*, 1878-86. *Lib. Com.* 1876-82.

1868 WILLIAMS, WILLIAM RHYS, M.D., Commissioner in Lunacy; 13, Gloucester street, Warwick square.

Elected

1863 WILSON, ROBERT JAMES, 7, Warrior square, St. Leonard's-on-Sea, Sussex.

1850 *WISE, ROBERT STANTON, M.D., Consulting Physician to the Southam Eye and Ear Infirmary; Beech Lawn, Banbury.

1825 WISE, THOMAS ALEXANDER, M.D., Thornton, Beulah Hill, Upper Norwood.

1879 WOAKES, EDWARD, M.D., Senior Aural Surgeon to the London Hospital; 78, Harley street, Cavendish square.

1885 WOLFENDEN, RICHARD NORRIS, M.D., Assistant Physician to the North-West London Hospital; 19, Upper Wimpole street.

1851 †WOOD, JOHN, F.R.S., Professor of Clinical Surgery in King's College, London, and Senior Surgeon to King's College Hospital; 61, Wimpole street, Cavendish square. C. 1867-8. V.P. 1877-8. *Referee*, 1871-6, 1880-86. *Lib. Com.* 1866. *Trans.* 3.

1848 †WOOD, WILLIAM, M.D., Physician to St. Luke's Hospital for Lunatics; 99, Harley street, Cavendish square. C. 1867-8. V.P. 1877-8.

1883 WOOD, WILLIAM EDWARD RAMSDEN, M.A., M.D., Rockhampton, Queensland.

1881 *WOODMAN, SAMUEL, Consulting Surgeon to the Ramsgate and St. Lawrence Royal Dispensary; 5, Prospect terrace, Ramsgate.

1879 WOODWARD, G. P. M., M.D., Deputy Surgeon-General; Sydney, New South Wales.

1878 YEO, GERALD FRANCIS, M.D., M.C., Professor of Physiology in King's College, London; Examiner in Physiology, University of London; King's College, Strand.

[It is particularly requested that any change of Title, Appointment, or Residence, may be communicated to the Secretaries before the 1st of October in each year, in order that the List may be made as correct as possible.]

HONORARY FELLOWS.

(Limited to Twelve.)

Elected

1847 CHADWICK, EDWIN, C.B., Corresponding Member of the Academy of Moral and Political Sciences of the Institute of France; Park Cottage, East Sheen.

1883 FRANKLAND, EDWARD, M.D., D.C.L., Ph.D., F.R.S., Corresponding Member of the French Institute; The Yews, Reigate Hill, Reigate.

1868 HOOKER, SIR JOSEPH DALTON, C.B., M.D., K.C.S.I., D.C.L., LL.D., F.R.S., Member of the Senate of the University of London, Director of the Royal Botanic Gardens, Kew; Corresponding Member of the Academy of Sciences of the Institute of France; The Camp, Sunningdale.

1868 HUXLEY, THOMAS HENRY, LL.D., D.C.L., F.R.S., Professor of Natural History in the Royal School of Mines; Secretary to the Royal Society; Corresponding Member of the Academies of Sciences of St. Petersburg, Berlin, Dresden, &c.; 4, Marlborough place, St. John's wood.

1878 LUBBOCK, SIR JOHN, Bart., M.P., D.C.L., LL.D., F.R.S., High Elms, Hayes, Kent.

1847 OWEN, SIR RICHARD, K.C.B., D.C.L., LL.D., F.R.S., late Superintendent of the Natural History Departments in the British Museum; Foreign Associate of the Academy of Sciences of the Institute of France; Sheen Lodge, Mortlake.

1883 PARKER, WILLIAM KITCHEN, F.R.S., Crowland, Trinity road, Upper Tooting.

Elected

1873 STOKES, GEORGE GABRIEL, M.A., D.C.L., LL.D., F.R.S., Lucasian Professor of Mathematics in the University of Cambridge; President of the Royal Society; Lensfield Cottage, Cambridge.

1868 TYNDALL, JOHN, D.C.L., LL.D., F.R.S., Professor of Natural Philosophy in the Royal Institution; Corresponding Member of the Academies and Societies of Sciences of Göttingen, Haarlem, Geneva, &c.; Royal Institution, Albemarle street, Piccadilly.

FOREIGN HONORARY FELLOWS.

(Limited to Twenty.)

Elected

1878 Baccelli, Guido, M.D., Professor of Medicine at Rome.

1883 Bigelow, Henry J., M.D., Professor of Surgery at Harvard University, and Surgeon to the Massachusetts General Hospital.

1876 Billroth, Theodor, M.D., Professor of Surgery in the University of Vienna; Vienna.

1883 Charcot, J. M., M.D., Physician to the Hôpital de la Salpêtrière, and Professor at the Faculty of Medicine of Paris; Member of the Academy of Medicine; Quai Malaquais 17, Paris.

1864 Donders, Franz Cornelius, M.D., LL.D., Professor of Physiology and Ophthalmology at the University of Utrecht.

1883 DuBois Reymond, Emil, M.D., Professor in Berlin; N. W. Neue Wilhelmstrasse 15, Berlin.

1866 Hannover, Adolph, M.D., Professor at Copenhagen.

1873 Helmholtz, Hermann Ludwig Ferdinand, Professor of Physics and Physiological Optics; Berlin.

1873 Hofmann, A. W., LL.D., Ph.D., Professor of Chemistry, Berlin.

1868 Kölliker, Albert, Professor of Anatomy in the University of Würzburg.

1856 Langenbeck, Bernhard, M.D., late Professor of Surgery in the University of Berlin.

Elected

1868 LARREY, HIPPOLYTE BARON, Member of the Institute of France; Inspector of the "Service de Santé Militaire," and Member of the "Conseil de Santé des Armées;" Commander of the Legion of Honour, &c.; Rue de Lille, 91, Paris.

1883 PASTEUR, LOUIS, LL.D., Member of the Institute of France (Academy of Sciences).

1878 SCANZONI, FRIEDREICH WILHELM VON, Royal Bavarian Privy Councillor, and Professor of Medicine in the University of Würzburg.

1856 VIRCHOW, RUDOLPH, M.D., LL.D., Professor of Pathological Anatomy in the University of Berlin; Corresponding Member of the Academy of Sciences of the Institute of France; Berlin.

LIST OF RESIDENT FELLOWS

ARRANGED ACCORDING TO

DATE OF ELECTION.

1833 Sir George Burrows, Bt., M.D., F.R.S.
Thomas A. Barker, M.D.
1835 Richard Quain, F.R.S.
Thomas A. Nelson, M.D.
1836 Alexander Shaw.
J. George French.
1837 Thomas Blizard Curling, F.R.S.
1838 Charles Hawkins.
Henry Spencer Smith.
1839 T. Graham Balfour, M.D., F.R.S.
Fred. Le Gros Clark, F.R.S.
James Dixon.
1840 Chas. J. B. Williams, M.D., F.R.S.
Charles Hutton, M.D.
Samuel A. Lane.
Sir James Paget, Bt., F.R.S.
1841 Sir Henry A. Pitman, M.D.
Sir William Bowman, Bart., F.R.S.
Paul Jackson.
1842 Charles West, M.D.
John Simon, C.B., F.R.S.
John Erichsen, F.R.S.
Sir Oscar M. P. Clayton.
1843 Robert Greenhalgh, M.D.
Sir Prescott G. Hewett, Bt., F.R.S.
Henry Lee.
Luther Holden.
Edward Newton.
1844 Arthur Farre, M.D., F.R.S.
William Wegg, M.D.
1844 Thomas King Chambers, M.D.
Edwin Humby.
1845 Samuel Cartwright.
George D. Pollock.
1845 Thomas Taylor.
Sir Edwin Saunders.
William Oliver Chalk.
Edward U. Berry.
Benjamin Ridge, M.D.
1846 John A. Bostock.
Barnard Wight Holt.
Carsten Holthouse.
1847 W. H. O. Sankey, M.D.
George Johnson, M.D., F.R.S.
1848 Sir Edward H Sieveking, M.D.
Edward Ballard, M.D.
William Wood, M.D.
Thomas Hawksley, M.D.
Edward John Tilt, M.D.
John Clarke, M.D.
John Gregory Forbes.
1849 Hugh J. Sanderson, M.D.
C. H. F. Routh, M.D.
Edmund L. Birkett, M.D.
George T. Fincham, M.D.
Sir William W. Gull, Bt., M.D., F.R.S.
1850 Richard Quain, M.D., F.R.S
George Roper, M.D.
1851 Sir Wm Jenner, Bt., M.D., F.R.S.
H. Haynes Walton.
John Birkett.
John A. Kingdon.
Peter Y. Gowland.
John Marshall, F.R.S.
John Wood, F.R.S.
Bernard E. Brodhurst.
Robert J. Spitta, M.D.
George Gaskoin.

1852 C. Bland Radcliffe, M.D.
Walter H. Walshe, M.D.
William Adams.
Sir Henry Thompson.
1853 Robert Brudenell Carter.
1854 Alfred Baring Garrod, M.D., F.R.S.
Samuel O. Habershon, M.D.
Sir Thomas Spencer Wells, Bt.
1855 W. M. Graily Hewitt, M.D.
J. Burdon Sanderson, M.D., F.R.S.
J. Russell Reynolds, M.D., F.R.S.
Walter John Bryant, M.D.
1856 Charles J. Hare, M.D.
William Bird.
Jonathan Hutchinson, F.R.S.
Timothy Holmes.
Alonzo H. Stocker, M.D.
1857 William Overend Priestley, M.D.
George Harley, M.D., F.R.S.
Henry Thompson, M.D.
Hermann Weber, M.D.
George Owen Rees, M.D., F.R.S.
John Whitaker Hulke, F.R.S.
John Morgan.
Henry Cooper Rose, M.D.
Henry Walter Kiallmark.
1858 Fred. George Reed, M.D.
William Chapman Begley, M.D.
John William Ogle, M.D.
Wilson Fox, M.D., F.R.S.
1859 Wm. Howship Dickinson, M.D.
William Scovell Savory, F.R.S.
Edwin Thomas Truman.
Richard Barwell.
Edward Tegart.
Septimus William Sibley.
William E. Stewart.
1860 Sir Andrew Clark, Bt., M.D., F.R.S.
William Ogle, M.D.
Thomas Bryant.
John Couper.
Henry Howard Hayward.
1861 Robert Barnes, M.D.
William Spencer Watson.
William Henry Holman, M.B.
1862 James Andrew, M.D.
Lionel Smith Beale, M.B., F.R.S.
Thomas H. Tuke, M.D.
Edmund Symes Thompson, M.D.
Reginald Edward Thompson, M.D.
William Henry Brace, M.D.
George Cowell.
Robert Farquharson, M.D., M.P.
M. Berkeley Hill.

1863 Octavius Sturges, M.D.
John Langdon H. Down, M.D.
Samuel Wilks, M.D., F.R.S.
Samuel Fenwick, M.D.
Julius Althaus, M.D.
Sydney Ringer, M.D., F.R.S.
Thomas Smith.
Arthur B. R. Myers.
Arthur E. Durham.
William Sedgwick.
1864 George Buchanan, M.D., F.R.S.
Charles Derby Waite, M.B.
John Harley M.D.
Walter John Coulson.
Thomas William Nunn.
Jos. Gillman Barratt, M.D.
1865 Charles Robert Drysdale, M.D.
James Edward Pollock, M.D.
William Cholmeley, M.D.
Reginald Southey, M.D.
George Fielding Blandford, M.D.
Sir Dyce Duckworth, M.D.
Frederick W. Pavy, M.D., F.R.S.
William Morrant Baker.
John Langton.
Frederick James Gant.
Alfred Willett.
Bowater John Vernon.
Alfred Cooper.
Christopher Heath.
1866 Thomas Fitz-Patrick, M.D.
Samuel Jones Gee, M.D.
Charles Theodore Williams, M.D.
Heywood Smith, M.D.
John Crockett Fish, M.D.
William Selby Church, M.D.
Edward John Waring, M.D.
1867 William Henry Day, M.D.
Achille Vintras, M.D.
Richard Douglas Powell, M.D.
F. Howard Marsh.
Henry Power.
Sir William MacCormac.
Thomas Pickering Pick.
John Astley Bloxam.
Charles Arthur Aikin.
Samuel Hill, M.D.
1868 H. Charlton Bastian, M.D., F.R.S.
William Henry Broadbent, M.D.
Thomas Buzzard, M.D.
John Cavafy, M.D.
Walter Butler Cheadle, M.D.
John Cockle, M.D.
Sir Thos. Crawford, K.C.B., M.D.

1868 T. Henry Green, M.D.
William Rhys Williams, M.D.
William Chapman Grigg, M.D.
John Croft.
George Eastes.
William Henry Freeman.
1869 Joseph Frank Payne, M.D.
Arthur E. Sansom, M.D.
John Wickham Legg, M.D.
Charles Elam, M.D.
Thomas Laurence Read.
1870 Alfred Meadows, M.D.
William Wadham, M.D.
J. Warrington Haward.
Edgcombe Venning.
Clement Godson, M.D.
1871 William Cayley, M.D.
Charles Henry Ralfe, M.D.
Arthur Julius Pollock, M.D.
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ADVERTISEMENT.

THE Council of the Royal Medical and Chirurgical Society deems it proper to state that the Society does not hold itself in any way responsible for the statements, reasonings, or opinions set forth in the various papers which, on grounds of general merit, are thought worthy of being published in its 'Transactions.'

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The Abstracts of the papers read will be furnished to the Journals as heretofore.

ADDRESS

OF

GEORGE JOHNSON, M.D., F.R.S.,

PRESIDENT,

AT THE

ANNUAL MEETING, MARCH 1st, 1886.

Gentlemen,—The preparation of the annual address with its obituary notices, at all times an anxious and a difficult task, has this year been rendered more than usually so by the fact that, unhappily, since the last anniversary meeting the number of our Fellows who have been taken from us by death is unusually large.

You will have learnt from the report of the Council that during the past year twenty-one Fellows of the Society have died. Of these six were *resident* Fellows, namely, Dr. Maclean, Mr. Arnott, Dr. Harris, Mr. John Gay, Dr. Wotton, and Dr. Sutro. Eleven were *non-resident* Fellows, namely, Dr. William Johnson Smith, Mr. Egerton, Dr. Livingston, Mr. Fortescue, Dr. Edward Howard, Dr. Wardell, Dr. James Russell, Dr. Scott, Mr. Tufnell, Mr. Page, and Dr. Maule Sutton.

To this list have to be added one *Honorary* Fellow, Dr. Carpenter, and three *Foreign Honorary* Fellows, namely, Professor Henle, Dr. Noël Gueneau de Mussy, and Professor Milne Edwards.

I propose now to refer to our deceased Fellows, resident and non-resident, in the order in which their deaths occurred, reserving for subsequent notice the names of the Honorary Fellows of the Society.

I have no doubt that each of my predecessors in this chair, while engaged in the responsible task of briefly sketching the lives and the professional work of those Fellows of the Society who had recently died, has, like myself, been influenced by the desire that his obituary notices should be animated by the same spirit of equity and of charity—equally remote from unmerited eulogy and from unfair criticism—as he would wish to be displayed by some future President when referring to his own professional career.

In preparing these biographical sketches I have derived much assistance from obituary notices which have appeared in the various public journals. In some instances, too, I am indebted to private friends and relatives of the deceased for information with which they have favoured me, and which I could not otherwise have obtained.

Dr. William Johnson Smith, of Weymouth, who was elected a Fellow of this Society in 1847, was born in October, 1813. He was educated in the University of Edinburgh, where he graduated M.D. in 1842.

In 1844 he became a member of the Royal College of Physicians, and afterwards settled at Weymouth, where he obtained a large practice. He there established the Weymouth Sanatorium for the treatment of diseases peculiar to women and children, which, from small beginnings, became in course of years a large and flourishing institution. In 1883 the friends of the Sanatorium placed in the entrance hall a marble bust of the founder, at a cost of £150. During the last two years of his life Dr. Smith suffered much from acute gout in his feet. He gradually became weaker, and died on the 12th of April, 1885, in his seventy-third year.

At his funeral, which was quite of a public character, a large number of friends and former patients attended to

pay their last tribute of respect and esteem for one whom they had learnt to look upon as a great public benefactor.

Mr. Charles Chandler Egerton[1] was born on the 13th of April, 1798, at his father's vicarage, Thorncombe, in Dorsetshire. Dr. Chandler, one of the physicians of Guy's Hospital, was his uncle, and Mr. Egerton received his medical education at the then united Guy's and St. Thomas's Hospitals, under Sir Astley Cooper, Mr. Travers, and others.

In May, 1823, he was appointed by the East India Company Assistant Surgeon on the Bengal establishment to practise as an oculist, and especially to take charge of the Lower Orphan School, composed of Indo-European lads who had contracted disease of the eyes; and at the end of the following month he sailed for Calcutta. Mr. Egerton dealt successfully with the epidemic in the Orphan School, and during his stay in India he held the first position as an oculist, first at the Eye Hospital, which was established under his own immediate care, and afterwards at the Medical College Hospital. He was a very skilful operator and a good surgeon.

He was appointed the first Surgeon at the Calcutta Medical College Hospital, and he held that appointment until he retired from the service. He had much influence in carrying out the plan of the Bengal Medical Retiring Fund when Lord Wm. Bentinck was Governor-General, and he assisted in the establishment of the Medical College for teaching the natives human anatomy by dissection.

Mr. Egerton left India at the end of 1846, or the beginning of 1847 and, having retired from practice, he went to live on his paternal estate, Kendal Lodge, Epping, where he died on the 4th of May last, at the age of eighty-seven. In 1858 he was placed on the Commission of the Peace for the county and until within five or six years of his death

[1] For the particulars of Mr. Egerton's work in India I am indebted to Dr. John Jackson, the well-known retired Indian practitioner.

he was one of the most regular attendants on the bench. One of his neighbours, Dr. Fowler, of Epping, who had known him for twenty years, says of him, in a note with which I have been favoured, "He was a man of no ordinary type; firm, resolute and self-relying, yet kind, hospitable, and benevolent. He was highly respected by his neighbours and by all who knew him, and warmly admired by his numerous friends." Mr. Egerton was elected a non-resident Fellow of this Society in 1823.

Dr. John Maclean was born at Shiels, near Renfrew, on the 13th of March, 1817. He was educated at the University of Glasgow and graduated M.D. in 1838. He became a member of the Royal College of Physicians in 1859, and was elected a Fellow of this Society in 1860. In 1845 he was appointed by the late Sir James Graham an Assistant Inspector of Prisons in the home district and, while holding this office, he was the author of numerous prison reports which were presented to both Houses of Parliament.

In 1847 Dr. Maclean was appointed Chief Medical Officer of the Mutual Provident Alliance Office, and in 1848 Physician to the Provident Life Office. His life office experience enabled Dr. Maclean to supply Mr. Gladstone, when Chancellor of the Exchequer, with statistics in aid of the Government scheme of life assurance. This service was acknowledged by Mr. Gladstone in his speech in the House of Commons, on introducing the Government Annuities and Assurance Bill in 1864.

Sir Spencer Wells, in a note with which he has favoured me, says that twenty years ago he often met Dr. Maclean on life assurance business, and he adds, "I was always impressed by the great care he devoted to this branch of the profession."

Dr. Maclean died on the 28th of April last, aged sixty-eight.

Mr. James Moncrieff Arnott[1] was born at Cupar-Fife on the 15th of March, 1794, where his father and his grandfather

[1] 'British Med. Journal,' June 20th, 1885.

had been in practice before him. He was educated, first at the grammar school of his native place and subsequently at the High School and the University of Edinburgh. He entered the medical classes in 1809, passed the Edinburgh College of Surgeons in 1813, and the following year obtained the M.D. of the University at the age of nineteen. Mr. Arnott then came to London for a year and attended Abernethy's lectures on anatomy at St. Bartholomew's and Astley Cooper's on surgery at Guy's. He also became a pupil at St. George's. In 1814 he went to Paris for a year, where he attended the classes of Pelletan and Dupuytren at the Hôtel Dieu and those of Roger and Roux at La Charité. He afterwards studied at Vienna for a year, chiefly under Beer, the ophthalmologist, and Hildebrand, the then famous teacher of clinical medicine. In 1817 Mr. Arnott returned to London and became a member of the Royal College of Surgeons. For many years he occupied himself by seeing the poor at his own house and often operating upon them at their homes. During these years he was a frequent visitor at the great hospitals on operation days.

At length, in 1831, Mr. Arnott was elected Assistant Surgeon to the Middlesex Hospital, and two years later he became full Surgeon. In 1836, while continuing to hold office as Surgeon at the Middlesex, he was appointed Professor of Surgery at King's College. This office he resigned in 1840, when, at the opening of the new King's College Hospital, he had to choose between the resignation of his Chair and that of his surgical appointment at the Middlesex. At that time his King's College pupils, of whom I was one, presented him with an illuminated address expressing their admiration of his character and his teaching and their extreme regret for his resignation.

In 1848 Mr. Arnott resigned his office at the Middlesex on being appointed Professor of Surgery at University College and Surgeon to University College Hospital. Two years later, in 1850, he retired from University College, and from that time he held no hospital appointment.

Mr. Arnott became a Fellow of the Royal College of

Surgeons in 1843, and an Examiner in 1847. He was twice elected President of the College—in 1850, and again in 1859. It was chiefly through his exertions that the College obtained the Government grant of £15,000 towards the rebuilding of the Hunterian Museum, and, aided by his former pupil, Mr. John Tomes, he did much to establish the license in dental surgery. In recognition of his services to the College, the Council, in 1852, voted the marble bust which may now be seen in the College.

He joined this Society in 1819, and since the death of Dr. Billing, five years ago, he had been our Senior Fellow. He held in succession nearly every office in the Society, and in 1847 he became President.

And here I am tempted to refer to a matter which occurred during his Presidency, his method of dealing with which serves, I think, to illustrate his good sense and discretion. In June, 1847, it happened that my friend and former colleague Mr. John Simon and I communicated each a separate paper on the same subject, namely, "Inflammation of the Kidney." The chief interest of the papers, and the only point of difference between the authors consisted in the interpretation of the microscopic appearances associated with the development of cysts in the kidney. The drawings which accompanied the papers were essentially alike, but the interpretation of the appearances by the respective authors was entirely different. In these circumstances, as I learnt afterwards from the President, some members of the Council suggested that both papers should be returned to the authors until they had found the means of reconciling their differences. Mr. Arnott, on the contrary, maintained that both papers should be published, together with their illustrations, so that facilities might be given for future observers to investigate the points in dispute. The President's arguments prevailed and the two papers, with their illustrative drawings, were published in the thirtieth volume of our 'Transactions.'

Mr. Arnott contributed eight papers to our 'Transactions;' of these the most important is entitled "A Patholo-

gical Inquiry into the Secondary Effects of Inflammation of the Veins." In this paper, which occupies 131 pages of the fifteenth volume of the 'Transactions,' after a full and complete reference to previous writers on the same subject, including not only English, but also French, German, and Italian authors, he gives a number of cases, and from the details of these he concludes that the fatal results of inflammation of the veins are due, not, as John Hunter had surmised, to the extension of the inflammation along the veins to the heart, but to the fact that the secondary abscesses in the viscera, the joints, and elsewhere are the result of contamination of the blood by pus and other morbid secretions. He insists on the resemblance between the secondary results of phlebitis and those diseases which are known to result from the inoculation of a morbid poison, and in this connection he makes especial reference to the local and constitutional symptoms which result from poisoned wounds received in dissection. And, lastly, he maintains that the secondary abscesses which sometimes result from injuries, whether of the extremities or of the head, and those which have not seldom followed parturition, have the same pathological origin, namely, the existence of phlebitis in the part of the body primarily affected, and the consequent transfer of infecting morbid materials to various remote parts.

Mr. Arnott was elected a Fellow of the Royal Society in 1843.

He held in succession various Royal appointments; he was Surgeon-Extraordinary to the late Queen Adelaide, Surgeon-in-Ordinary to the late Prince Consort, and Surgeon-Extraordinary to the Queen. In 1865 he retired from active practice on succeeding to an old family estate at Chapel in Fifeshire.

During the last two years of his life, Mr. Arnott occasionally asked me to see him on account of some disturbance of the circulation which was associated with evidence of atheromatous degeneration of the arteries and with a loud systolic murmur over the apex of the heart. In the

early part of last year his only daughter, who was his constant companion, noticed that he was losing colour and strength, and when he came to London in the spring, Mr. Sibley and I were asked to consult together upon his condition. We found him greatly changed in appearance, without discoverable organic disease, other than the state of the circulation before mentioned. He continued to lose flesh and colour until he was suddenly seized with urgent dyspnœa and extreme restlessness, symptoms which led us to the conclusion that a clot in the right side of the heart or in the pulmonary artery was obstructing the flow of blood through the lungs. After a few hours of acute suffering he died on the 27th of May in the ninety-second year of his age.

His funeral in Kensal Green was attended by Mr. Cooper Forster, then President of the Royal College of Surgeons, and by many friends.

Mr. Arnott was universally held in the highest esteem not only for his acknowledged professional skill and acquirements, but also for his unswerving integrity. I can bear personal testimony to the high appreciation of his clear and emphatic teaching by those who attended his lectures.

I remember once being much impressed, in common with my fellow-students, by the candid manner in which he acknowledged an error of diagnosis. We had gone to the Middlesex Hospital to see him operate; and a testicle believed to be medullary was removed. After the patient had been carried out, Mr. Arnott sliced the testicle, and turning at once to the class, without a moment's delay or hesitation, he said, "Gentlemen, we have been mistaken; that which we took for malignant disease of the testicle we now find to be a hæmatocele."

Mr. George Fortescue[1] was a native of Cornwall, and in 1840, when scarcely two years of age, was taken by his parents to Tasmania, where, at Christ's College, he

[1] 'Australian Medical Gazette,' June 15th, 1885.

received his primary education, and subsequently he returned to complete his education in England.

In 1857 he entered the Medical School of King's College, where in 1858 he obtained a junior scholarship, in 1859 a prize in Chemistry, and in 1861 he was appointed House Surgeon. He was a general favourite amongst his contemporaries, and was greatly admired for his splendid physique. The museum of King's College contains a cast of his right arm, displaying a magnificent muscular development, and there is a tradition that on one occasion a fellow-student, having insulted him, was seized and held at arm's-length over the baluster of the hospital staircase, with a threat that if the offence were repeated he should be dropped upon the pavement below. Having obtained the M.R.C.S. in 1860, and graduated M.B. London in 1861, he soon afterwards returned to Australia, and for near a quarter of a century he was one of the leading practitioners of Sydney. For many years he was Surgeon of the Sydney Infirmary, and subsequently Surgeon of the Prince Alfred Hospital, from its foundation to the time of his death, which occurred on the Paramatta River near Sydney on the 1st of June, 1885, at the age of forty-seven, from an attack of typhoid fever.

Mr. Fortescue was highly esteemed in the community amongst whom he had lived and worked. Respected for his skill in the profession he for so many years adorned, he was no less beloved in private life, for the many kindly and genial qualities he possessed. His own saying that absolute "sanity" is the highest human quality, is said to have been thoroughly exemplified in his character. He was elected a Fellow of this Society in 1877.

Dr. John Livingston, whose death at the age of forty-five occurred suddenly from apoplexy on the 10th of June last, was educated at the University of Glasgow, where he graduated M.D. in 1861. For a number of years Dr. Livingston had a large practice at New Barnet, where I have occasionally met him in consultation, and was much impressed by his intelligence and his energy. Amongst

other appointments he held that of Medical Officer of the Great Northern Railway. Dr. Livingston was elected a Fellow of this Society in 1870.

Dr. Edward Howard was M.R.C.S. 1838, L.S.A. 1839, M.D. Giessen, 1844, M.R.C.S. London, 1860.

He was appointed Assistant Surgeon in the 20th Regiment of Foot in 1842. He became Surgeon in 1854, and Surgeon-Major in 1862. In 1867 he retired on half-pay with the honorary rank of Deputy Inspector-General. For more than twenty years Dr. Howard was on foreign service in various parts, Bermuda, Canada, Turkey, and the East Indies. For his services in Turkey he received the Order of the Medjedie (5th Class). The Director-General of the Medical Department of the Army, to whom I am indebted for the particulars of Dr. Howard's services, states that "this officer was highly esteemed by his brother officers, and his duties were always performed to the satisfaction of the Director-General."

I learn from Dr. Goldsmith, who had attended Dr. Howard for many years, that he caught a terribly severe epileptiform neuralgia in the trenches before Sebastopol, and that this malady clung to him for the remainder of his life. He died at Bedford on the 28th June of last at the age of sixty-nine. He was elected a Fellow of this Society in 1865.

Dr. John Richard Wardell[1] was born at Pickering in Yorkshire in September, 1819. After receiving his early education at a private school in Doncaster he began the study of Medicine in the University of Edinburgh, where he graduated M.D. in 1844. During his residence in Edinburgh he filled the offices of Assistant Pathologist and Resident Physician at the Royal Infirmary. He was also President of the Royal Physical and Hunterian Societies. In 1859 he became a Member of the Royal College of Physicians, and in 1867 he was elected a Fellow of the College. He was elected a Fellow of this Society in 1858.

During the earlier part of his professional life Dr.

[1] 'British Medical Journal,' Sept. 6th, 1885

Wardell acted as private physician to a gentleman of rank, upon whose decease he commenced practice at Tunbridge Wells. There until within four years of his death he continued to practise, and was acknowledged as the chief consultant of the town and neighbourhood. As Physician to the local Infirmary he devoted much time to laborious and careful clinical research, the good results of which are apparent in his numerous professional writings. Four years ago he was struck down by illness and compelled to relinquish practice. He went for rest and change to Brighton, where for a time he was restored to a moderate state of health, but a few days before his decease the symptoms became aggravated, and he died on the 21st of August. Throughout his prolonged illness his mind remained clear and active, and during the last year of his life he collected and published in a large octavo volume of 800 pages entitled 'Contributions to Pathology and the Practice of Medicine,' some of his numerous and varied professional writings. The volume consists of fifty chapters on a great variety of subjects, affording conclusive evidence of great industry, extensive reading, careful clinical observation, close and accurate reasoning and great practical skill in the prevention and treatment of disease. The longest and most elaborate chapter is that on relapsing fever, which is based on the author's observation of that disease in Edinburgh during the epidemic of 1842-3, and which, as he says, he was induced to republish mainly by a remembrance of the value which the late Dr. Murchison put upon the facts and statistics there given. One of the most interesting and instructive chapters in the book is that entitled "A Thorn in the Flesh," in which the author gives a graphic account of his own prolonged and severe suffering from inflammation and abscess in the lower part of the thigh, by which the loss of the limb was threatened, and which was ultimately found to have been caused by a thorn, an inch and a half long, which he concluded must have penetrated the thigh five years before, when his horse fell in

leaping a hedge. The removal of the foreign body was at length followed by a complete cure.

Dr. Francis Harris[1] was born on December 1st, 1829, at Winchester Place, in Southwark. His father, who had for some time represented the borough in Parliament, died while the son was very young. After his earliest schooling and some later studies at King's College, London, he entered at Caius College, Cambridge. He graduated B.A. in 1852. After leaving Cambridge he entered as a student at St. Bartholomew's. He graduated M.B. in 1854. From November, 1856, to August, 1857, he was House-Surgeon to the Hospital for Sick Children in Great Ormond Street. In 1857 he was admitted M.R.C.P. London. In the same year he went to Paris for six months and afterwards to Berlin, where he attended Virchow's lectures, and he subsequently visited Saxon Switzerland, Dresden, Prague, and Vienna in company with Dr. Chance. Returning to England after an absence of about a year, he was appointed Demonstrator of Morbid Anatomy at St. Bartholomew's; he was also elected Obstetric Physician to the St. George's and St. James's Dispensary, and Assistant Physician to the Hospital for Sick Children in May, 1859. The same year he took his degree of M.D., and chose for his academical disputation "The Nature of the Substance found in the Amyloid Degeneration of Various Organs in the Human Body." This essay, which was printed in 1860, was his only published work. He was elected a Fellow of the College of Physicians in 1863. The dispensary he soon gave up and with it any intention he may have had of practising obstetrics. After Dr. Baly's accidental death in 1861 Dr. Harris was elected Assistant Physician to St. Bartholomew's and, about the same time, he was appointed Lecturer on Botany, a science in which he took a deep interest to the end of his life. In 1865 he resigned the Children's Hospital and the Lectureship on Botany, and bought an estate which was situated partly

[1] For the particulars of Dr. Harris's career I am indebted to a memoir by Dr. Gee, in the 'St. Bartholomew's Hospital Reports,' vol. xxi.

in Lamberhurst and partly in Brenchly parish, in the Weald of Kent. His love of a country life drew him more and more away from London and from the pursuit of his profession. In 1863 he was elected Physician to St. Bartholomew's. At that time he had retired from all medical work except at the hospital, and he lived as much as possible on his estate, taking especial pleasure in his garden, his orchard house, his vinery, and latterly in his orchid houses, where he turned his botanical knowledge to good account and made numerous successful experiments in crossing orchids.

In 1874 ill-health compelled him to resign his hospital duties. Two or three years before this time he began to suffer from progressive emphysema and pulmonary catarrh connected with a disposition to gout, and these infirmities gained upon him somewhat quickly. During the last three or four years of his life dyspnœa was almost continual and sometimes very severe. In June, 1882, he had an attack of pneumonia, and a recurrence of this disease put an end to his life on September 3rd, 1885. His death was felt to be a great loss by many friends both in town and country, to whom his kind and hospitable spirit had made him dear.

One friend and former pupil (Dr. Andrew) bears testimony to Dr. Harris's high qualities and success as a teacher of pathological anatomy,—"the severity of study being relieved by his ready wit and sense of humour." Another friend (Dr. Chance) says, "That he might have made a large practice is undoubted. His presence was good and calculated to inspire confidence. All that he wanted was energy, ambition, and lack of money. If he had had no money he would have made it; but even then he would have stopped when he thought he had sufficient." Dr. Chance adds, "I used to go to him not only for the sake of his conversation, but to ask him for advice, for I considered his judgment to be very sound."

Mr. John Gay[1] was born at Wellington, Somerset, in

[1] 'Lancet' and 'British Medical Journal,' Sept. 26th, 1885.

September, 1813, and began the study of his profession under the late Mr. Bridge in his native town. In 1833 he entered at St. Bartholomew's, where he was clinical clerk to Dr. Latham and dresser to Sir William Lawrence, and where he was at the head of the prize list. In 1834 he became a Member of the Royal College of Surgeons, and in 1843 an Honorary Fellow. In 1836 he was elected Surgeon to the Royal Free Hospital, an appointment which he held with great credit to himself and advantage to that institution until the year 1853, when he became Senior Surgeon to the Great Northern Hospital, an appointment which he continued to hold during the remainder of his life.

Mr. Gay obtained a considerable practice in the City, and he was the author of various original and important contributions to the science and practice of surgery. Of these one of the earliest and most valuable was a treatise 'On the Anatomy, Pathology, and Surgery of Femoral Hernia," published in 1848. The main object of the author was to deprecate too free incisions into the hernial sac, by which not only is the immediate risk of the operation greatly increased but a future return of the hernia is rendered probable. The principles of Mr. Gay's operation "consisted in reaching the seat of stricture when external to the sac by a small incision made through healthy structures and in such a situation that the hernial mass shall not be injured or disturbed thereby." Sir William Fergusson said of this proposal, "By this simple difference a vast improvement has been effected in the operation for crural hernia."

In 1855 Mr. Gay published 'A Memoir on Indolent Ulcers and their Surgical Treatment.' In this treatise he advocated the practice of making free incisions through the indurated tissues, the object being to relieve tension and so to favour cicatrisation. The practice is said to be good and successful.

In the Lettsomian Lectures delivered at the Medical Society of London in 1867-8 and subsequently published, Mr. Gay discussed the treatment of varicose veins and allied

disorders. He maintained that the common practice of treating this troublesome condition by prolonged rest and permanent bandages tends to increase congestion of the skin and the subcutaneous tissues, and to cause an injurious dilatation of the deeper veins. The lectures were illustrated by numerous elaborate dissections.

Mr. Gay's last contribution to surgical literature was a paper "On certain points connected with the Anatomy of the Venous System," which was read before the Medical Society of London in November, 1883. In addition to the publications before mentioned, Mr. Gay from time to time communicated to the medical societies and to the medical journals papers of high practical value on various important points in surgery.

In 1869 Mr. Gay was elected a Member of the Council of the Royal College of Surgeons. In 1877, when his term had expired, he failed to secure his re-election, but in the following year he was successful.

He joined this Society in 1848 and served on the Council in 1874-5.

In the autumn of 1883 Mr. Gay had an attack of hemiplegia. From this illness he never recovered, and for some months before his death, to the distress of his family and numerous friends, he remained in a condition of semi-consciousness. At length he died tranquilly on the 15th of September, 1885, in the seventy-second year of his age.

Mr. Gay had a large circle of friends both in and beyond the limits of his profession. He was held in the highest esteem not only on account of his honorable and successful surgical career, but his bright intellect, his varied accomplishments, and his admirable social qualities endeared him to all his intimate associates.

Dr. James Russell, who was a descendant of one of the oldest and most influential Nonconformist families of Birmingham, was born in that city on the 1st of April, 1818. His father practised in New Hall Street, Birmingham, for more than half a century, and was highly esteemed as an able practitioner, and a most conscientious and benevolent

man. His great-uncle, William Russell, of Showell Green, was one of the Nonconformists whose houses were pillaged and burnt during the disgraceful Church and King Riots in 1791, at the same time that the philosophic Priestley was driven from the town.

James Russell received his early education under the Rev. E. Bristowe, and in addition he took mathematical lessons from the Rev. W. Lawson, of Moseley.

In 1835 he entered at the then newly-established "School of Medicine," now known as Queen's College, whence in 1840 he removed to King's College, London, where I made his acquaintance, which led to a lifelong friendship. His choice of King's College as a school, notwithstanding his staunch Nonconformist principles, was doubtless in great part determined by the fact that three distinguished Birmingham men, and more or less intimate friends of himself and his father, were then on the teaching staff of the College. Mr., now Sir William, Bowman, was Demonstrator of Anatomy and Assistant Surgeon to the hospital, the late Mr. Partridge was Professor of Anatomy and Surgeon to the Hospital, and the late Dr. William Allen Miller, while pursuing his medical studies with a view of obtaining the M.D. of London, was acting as Assistant to the late Professor Daniell, whom he afterwards succeeded in the Chair of Chemistry.

During his pupilage at King's College James Russell was held in the highest esteem, both by his teachers and by his fellow-students, amongst whom his irreproachable character, his great intelligence, his untiring industry and devotion to duty, his unswerving truthfulness, and, in spite of an occasional combativeness in argument and brusqueness of manner, his genuine kindness of heart and his tolerance of diverse opinions, were thoroughly and very generally appreciated.

At the end of his student career he held, for the usual period of six months, the office of House Physician of the hospital, and during this period I had the privilege of being his colleague as House Surgeon.

He passed what is now called the Intermediate, and the M.B. examination at the University of London in the same year, 1842, and at the latter examination he was second in the list of honours in surgery. He graduated M.D. in the first division, in 1848.

Originally intending to practise surgery he was elected one of the Honorary Surgeons of the Birmingham General Dispensary in 1844, but he was soon induced to change his views, and in three months, having resigned his surgical appointment, he henceforth devoted himself entirely to the study and practice of medicine; and as a preparation for practising as a physician he went to Paris and pursued his studies there for a considerable period. On his return in 1847 he commenced practice in Temple Row. He became a Member of the Royal College of Physicians in 1859, and in 1867 he received the well-deserved honour of the Fellowship.

In 1848 he was elected Honorary Physician to the General Dispensary, an appointment which he held for five years.

In 1850, when the Sydenham College Medical School was established, Dr. Russell was appointed Lecturer on Therapeutics in the Materia Medica course, a position which he occupied with marked success for a period of sixteen years. He then joined Dr. Bell Fletcher as co-lecturer on the Practice of Physic, of which subject he retained the Professorship after the amalgamation between the Sydenham and Queen's Colleges had been accomplished.

In 1859 Dr. Russell was elected one of the Physicians of the General Hospital, where one of his former colleagues (Mr. Alfred Baker) says of him:—"His painstaking interest in the regular instruction of students in attendance was on a par with his unflagging attention to the wants and comforts of the sick. His hospital labours were assiduous and thoughtful, contributing to the stability, high character, and popularity of the Institution. The medical periodicals testify to his research, his accuracy of observation, his diagnostic skill, and his cautious conclusions;

qualities that are very notable in his comments on intricate nervous maladies, which were always interesting subjects of his study."

At the commencement of last year failing health compelled him to resign his hospital appointment, when his past and present pupils, to the number of 109, subscribed to a testimonial fund, and the subscribers and friends of the hospital commissioned Mr. Papworth to execute a marble bust.

Dr. Russell, as a townsman, was a steady supporter of all educational movements and of all public sanitary measures. He also devoted much time to the management of various charities. His nomination as a borough magistrate in 1880 gave satisfaction alike to the profession and the public.

About a year before his death Dr. Russell discovered that he was the subject of a serious form of Bright's disease, and, with a full knowledge of what this involved, he, for a time, kept almost complete silence on the subject —confiding the fact only to one or two of those from whom it was not prudent and scarcely possible to conceal it—his object being to prevent the lives of others from being darkened by the cloud of sorrow before the stern necessity arose. He suffered much during the last months of his life from that distressing form of dyspnœa which so often results from the later stages of the disease, but his intellect remained unclouded until the last. At length on the 5th of October, 1885, he was released from suffering.

Of all the men whose friendship I have had the privilege of enjoying, I know of no one who appeared to me to act more consistently upon the maxim, "Whatsoever thy hand findeth to do, do it with thy might," than Dr. James Russell, who since the year 1845 had been a Fellow of this Society.

Mr. Thomas Jolliffe Tufnell,[1] the well-known Dublin Surgeon, was a younger son of Colonel Tufnell, of Lachlam House, Chippenham, Wilts, where he was born

[1] 'Lancet' and 'Medical Times and Gazette,' Dec. 5th, 1885.

in 1819. In 1836 he was apprenticed to Mr. Limscombe, of Exeter, and subsequently entered at St. George's Hospital. In 1841 he became a Member of the College of Surgeons, and soon after entered the Army as Assistant Surgeon of the 44th Regiment, which was then serving in India. On his arrival at Calcutta to join his regiment he was ordered to take charge of the troops at Chinsura, and thus he escaped the massacre of the British forces in the disastrous retreat from Cabul. On his return home he was appointed Surgeon to the Dublin District Military Prison. When the Crimean War broke out Mr. Tufnell again went on foreign service, and during that campaign he obtained an extensive practical knowledge of military surgery. After his return to Dublin he retired from active service, and was appointed Surgeon to the City of Dublin Hospital; and when, after many years, he resigned the office of Visiting Surgeon, he was unanimously elected Consulting Surgeon to the Hospital. He was for some years Professor of Military Surgery in the School of the College of Surgeons, and also an Examiner in that institution. In the year 1873 he was elected Vice-President, and the following year President of the Dublin Royal College of Surgeons.

Mr. Tufnell was the author of several monographs on surgical subjects. Of these, the earliest was entitled 'Practical Remarks on the Treatment of Aneurism by Compression,' 1851. In 1864 he was elected a Fellow of this Society, and in 1873 he communicated a paper, which is published in the 57th vol. of the 'Transactions,' "On the Successful Treatment of Aneurism by Position and Restricted Diet." This paper contains the history of two cases of aneurism of the abdominal aorta and one of popliteal aneurism, in each of which a cure was effected. These cases are republished, with coloured illustrations, in the author's treatise on 'The Successful Treatment of Internal Aneurism by Consolidation of the Contents of the Sac,' 2nd edition, 1875. In one of the cases of cured abdominal aneurism (that of John Kelly, pp. 29 to

34) the patient is reported to have died some weeks afterwards of Bright's disease. But the excellent coloured illustration which accompanies the case shows, I think, that the different morbid conditions of the two kidneys were not due to Bright's disease, but were an indirect result of the aneurism which implicated the aorta at the place of origin of the renal arteries. The right kidney was "rather smaller than natural," and has obviously been invaded by embolic particles of fibrine from the interior of the aneurism. The left kidney, on the other hand, was "greatly enlarged, measuring five inches in length and three and a half inches in width." The renal veins are not represented in the drawing nor is their condition described, but there can, I think, be no doubt that the structural changes in the enlarged left kidney were caused by compression of the vein in its passage over the large aneurism towards the vena cava. Although, therefore, the aneurism was filled by firm fibrinous coagula, the cure was not effected before serious structural changes had occurred in both kidneys, but more especially in the left.

In 1879 Mr. Tufnell published a paper on "The Consolidation of Internal Aneurism," in which he rightly maintained, in opposition to Dr. William Colles, that the fibrinous layers within an aneurismal sac are the result of successive deposits from the blood, and not an exudation from the walls of the aneurism.

Amongst other papers by the same author may be mentioned one "On Luxation Downwards and Backwards of the three Internal Metatarsal Bones, a form of Dislocation of the Foot not previously described," 1854. "Practical Remarks upon Stricture of the Rectum, especially in relation to its connexion with Fistula in Ano and Ulceration of the Bowel," 1860. "On the Radical Cure of Varicocele by Subcutaneous Ligature of the Spermatic Veins" from the 'Dublin Journal.'

Mr. Tufnell died on the 27th of November last after a tedious illness at the age of sixty-seven. He was highly

esteemed by all classes, not only for his professional abilities and attainments, but also for his upright and honorable character and his kind and courteous disposition.

Dr. John Moore Johnston Scott[1] was born in Belfast, December 4th, 1850. He passed his matriculation examination and commenced his medical studies in Queen's College of his native city in 1869, where he is said to have secured the esteem and affection of his fellow-students.

After the breaking out of the Franco-German war, although he had not yet completed his full course of study, he was induced by a love of adventure and a desire to increase his professional knowledge and experience, to apply for, and through the interest of Sir William Mac Cormac, he obtained, the appointment of Assistant Surgeon to the Anglo-American Ambulance Corps. In this capacity he worked with his corps in aid of the French troops at Sedan. For his services during the war he received a bronze medal and a flattering testimonial from the French Government. After returning home he resumed his studies, and in 1842 he passed his examination in medicine, surgery, and obstetrics, and graduated M.D. in the Queen's University.

Soon after this he commenced practice in Belfast where he was highly successful. But in 1878 an eligible opening having occurred in Lurgan, Co. Armagh, Dr. Scott determined to take advantage of it. There his genial disposition gained for him an early and hearty admission to the good graces of all classes and creeds of his fellow-townsmen. Though a prominent Conservative and an energetic Orangeman, he never allowed his political or his religious opinions to interfere with his private relations or his professional duties.

In 1881 his popularity was shown by his return at the head of the poll as a candidate for a seat at the local Municipal Board. In 1882 he was elected a Guardian

[1] 'Lurgan Times,' Dec. 5th, 1885.

of the Lurgan Union, and in that position his exertions on behalf of both the ratepayers and the poor were unceasing and well-directed.

Dr. Scott, though to outward appearance in robust health, had for some time been aware that his heart was unsound, and on the 30th of November last, which was the day appointed for the parliamentary election in Lurgan, while conversing in the street with some friends on the prospects of the election, he suddenly staggered and fell backwards, his head, however, not coming in contact with the ground. He was immediately carried into a neighbouring office, where he retained consciousness until the arrival of Dr. Adamson, who happened to be near the spot, and whom he requested to examine his heart. In a few minutes, however, the pulse and breathing had ceased.

At his funeral, although a hearse had been procured, his brethren, the Town Commissioners, insisted on carrying the coffin to the grave; and, notwithstanding the inclemency of the weather, his fellow-townsmen of all classes assembled to pay the last tribute of respect to one whom they had learned to regard with feelings of the closest personal attachment.

Dr. Scott had been a Fellow of the Society since 1873.

Dr. Henry Wotton received his medical education at University College. He became a Member of the Royal College of Surgeons in 1859, and a Fellow by examination in 1864. He was elected a Fellow of this Society in 1865. In 1878 he graduated M.D. at St. Andrews.

He was Surgeon-Accoucheur to the West London Lying-In Institution, and he practised at Kensington, where he died suddenly on Christmas Day last at the age of forty-six. The verdict of the coroner's jury was "Suicide during temporary insanity." Such a catastrophe as we know may overtake the wisest and the best of men.

> "This frail bark of ours, when sorely tried,
> May wreck itself without the pilot's guilt,
> Without the captain's knowledge."
>
> TENNYSON, "*Aylmer's Field.*"

Mr. William Bousfield Page,[1] who died at St. Ann's, Carlisle, in his sixty-ninth year, on the 5th of January last, was born at Ashford in Kent in the year 1817.

He belonged to an Essex family, who have long had their seat at Southminster Hall, where they still reside. He received his medical education at the London Hospital, became a Member of the College of Surgeons and of the Apothecaries' Society in 1841, and a Fellow of the College in 1856. At the early age of twenty-four, on the recommendation of Mr. John Scott, then one of the Surgeons of the hospital, Mr. Page was appointed Surgeon to the Cumberland Infirmary, which had been recently established. He arrived in Carlisle on New Year's Day, 1843, an entire stranger to the city, but being possessed of courage and tact, as well as skill, he set to work with great energy and soon found many influential friends. He had not been three days in the city before he was summoned to attend a member of the Bishop's family, and in the course of a few years he became the trusted adviser of all the cathedral dignitaries and of the leading county families. During the London season he had so many of his county patients here that he had serious thoughts of settling in the metropolis; notably in 1851, when Sir B. Brodie advised him to apply for the appointment of Surgeon to the then recently opened St. Mary's Hospital. This appointment, however, he left for his eldest son at a later period to obtain.

Mr. Page rendered important services to several of the great railway companies. In this service his promptness and his organising power had full play, and in the distressing scenes of a great accident his self-possession and his skilfully applied surgical resources animated all around.

With regard to subsequent claims for compensation his advice, which was always implicitly relied upon, often resulted in an equitable arrangement without resort to costly and uncertain legal proceedings.

[1] The 'Carlisle Patriot,' Jan. 8th and 15th, 1886; 'Lancet,' Jan. 23rd, 1886.

In connection with his work at the Infirmary, Mr. Page induced Bishop Percy to institute a system of boarding out convalescents, which in time resulted in the establishment of the Sanatorium at Silloth. He was also the prime mover in the measures which led to the enlargement of the Infirmary, which now contains 100 beds, one of the wards, in well-deserved compliment to him, being named "The Page Ward."

In 1877 he resigned the office of Surgeon to the Infirmary, when he received a cordial vote of thanks for his distinguished services, and at the same time he was appointed Consulting Surgeon and a Vice-President.

Among other public appointments Mr. Page was for many years Surgeon to the Gaol and Consulting Surgeon to the Lunatic Asylum. In 1877 he resigned his office in the Gaol, and at the ensuing Quarter Sessions he received a cordial vote of thanks for his valuable services to the county and for his disinterestedness in relinquishing his right to a pension.

For more than a quarter of a century Mr. Page was a Justice of the City of Carlisle, and in 1878 he was appointed a Magistrate for the County of Cumberland. Apart from his profession he took a lively interest in all local works of public benefit, and he was always a wise and munificent supporter of charities.

He was elected a Fellow of this Society in 1847, and he contributed two papers to the 'Transactions,' one on "Cases of Ununited Fracture successfully treated" (vol. xxxi), and the other "On Excision of the Os Calcis in Incurable Disease of the Bone as a substitute for Amputation of the foot" (vol. xxxiii). In the earlier years of his practice he contributed various papers to the medical journals.

He was a bold and successful operator. The 'Lancet' of April 5th, 1845, contains the first account of his success as an ovariotomist, and as long ago as 1846 he had obtained complete success in two cases of excision of the knee-joint.

Mr. Page had been in good health until within nine

months of his death, when his strength began and continued to fail from a progressive anæmia, the starting-point of which seemed to be the shock of a heavy personal sorrow.

The large and distinguished assembly at his funeral, including the bishop of the diocese, who took part in the service, afforded a striking demonstration of the high estimation in which he was held by those who were best able to appreciate his character and his public services.

It is a remarkable circumstance that within forty-eight hours of Mr. Page's death his only brother died, after a short illness, and the two brothers were buried together.

Dr. John Maule Sutton,[1] who was born in 1829, was a great grandson of Mr. Daniel Sutton,[2] the famous inoculator for small-pox in the last century, to whom in 1767 King George III granted a patent of arms.

Dr. Sutton, having when young been left an orphan, was educated under the care of his grandfather, the late Mr. John Sutton, of Lee, Kent. He received his medical education at Queen's College, Birmingham, and at St. Thomas's Hospital.

Amongst other legal qualifications he obtained the following: F.R.C.P. Edin., 1853; M.R.C.P. Lond., 1859; M.D. St. And., 1853; M.R.C.L. Eng., 1851; L.M., 1853; L.S.A., 1853. He must therefore have had a full share of medical examinations.

Dr. Sutton, after serving the office of Resident Physicians' Assistant at the Brompton Hospital for Consumption, commenced practice in Bath, and was elected Physician to the Eastern Dispensary, and on resigning the appointment to take up his residence in Pembrokeshire—where some landed property had come into his possession—he was made a Life Governor in recognition of his services. Having settled at Tenby he devoted himself assiduously

[1] For the particulars of Dr. Sutton's career I am indebted to Mr. Joseph Chambers, chief clerk in the Officer of Health's Department, Oldham.

[2] "The Inoculator or Suttonian System of Inoculation,' by Daniel Sutton, Surgeon, 1796; 'The Tryal of Mr. Daniel Sutton for the High Crime of preserving the lives of His Majesty's Subjects by Inoculation,' 2nd ed., 1767.

to his profession, and took a prominent part in public affairs. He was three times elected mayor of Tenby, and subsequently he was appointed a Justice of the Peace for the Borough of Tenby and for the County of Pembroke. He also became Deputy-Lieutenant of the county.

In 1863 he was elected Physician to the Queen's Hospital, Birmingham, and Professor of Clinical Medicine. About that time the inhabitants of Pembrokeshire presented him with a service of plate at a public dinner, which was presided over by Captain Ramsay, R.N., C.B., afterwards Earl of Dalhousie, the father of the present earl.

In 1865, on the death of his aged grandfather, under whose will he obtained an increase of fortune, Dr. Sutton, who had never taken up his residence in Birmingham, resigned his appointment at the Queen's Hospital and retired from private practice.

Soon after this he invested largely in a colliery yielding "anthracite coal," and as the colliery not long afterwards became flooded and had to be abandoned, he thereby lost the greater portion of his fortune.

In 1873 Dr. Sutton was appointed the first Medical Officer of Health, under the Public Health Act, for the Borough of Oldham. There he organised most thoroughly the Sanitary Department, and soon, by his genial disposition, drew round him a host of friends and supporters.

In 1877 an epidemic of smallpox having broken out, he made it the occasion for founding the Westholme Hospital for Infectious Diseases, which, having been subsequently enlarged, now contains 100 beds.

Dr. Sutton during his ten years' tenure of office is said to have treated in that hospital upwards of 600 patients with great care, skill, and kindness.

The subject of infant mortality was one to which he devoted much attention, and upon which he published a treatise, and another on "Day Nurseries and their bearing upon Public Health."

Dr. Sutton devoted much attention to the means of

abating the "Smoke Nuisance." He contributed to the 'Lancet' (1871) a paper on the "Deodorisation and Utilisation of Town Sewage." He founded a Meteorological Observatory in the Alexandra Park, Oldham, where during the last ten years observations have been regularly taken; and as a Governor of the Oldham Infirmary he established the Hospital Saturday Collection, from which the Institution now receives a considerable annual sum.

After ten years of great public service to the borough ill-health compelled Dr. Sutton to resign his office in September, 1883. He then went to reside at Hoylake, a small village on the coast of Cheshire, where he died from disease of the heart with dropsy on the 20th of January last, at the age of fifty-six. He has left a large circle of sorrowing friends.

Dr. Sutton was elected a Non-Resident Fellow in 1855.

Dr. Sigismund Sutro was born in Bavaria in 1815. He studied medicine at Heidelberg and Munich, and at the latter University he obtained his degree of Doctor of Medicine in 1840. Soon after this he came to London, and in 1845 he was appointed Physician to the German Hospital when that institution was in its infancy. He resigned that office in 1877, and was then appointed Consulting Physician.

In 1859 Dr. Sutro became a Member of the College of Physicians, and in 1873 he was elected a Fellow. He had a considerable knowledge of the Spas of Europe, and especially those of Germany, and he was the author of a practical work on the subject, a second edition of which was published in 1865. Dr. Sutro's advice was highly valued by his countrymen, and especially by his co-religionists of the Jewish persuasion. He was in active practice up to the time of his death, which occurred on the 19th of February from an attack of apoplexy.

Dr. Sutro was elected a Fellow of this Society in 1860.

Dr. William Benjamin Carpenter[1] was born at Bristol in

[1] 'Times,' Nov. 11th, 1885; 'Lancet' and 'Brit. Med. Jour.,' Nov. 14th.

1813. He was the son of Dr. Lant Carpenter, an eminent Unitarian minister, under whose superintendence the son was educated. Dr. Carpenter's medical education was commenced at Bristol, but at the age of twenty he entered the Medical School of University College. He became a Member of the College of Surgeons and a Licentiate of the Apothecaries' Company in 1835, after which he went to Edinburgh, where he graduated M.D. in 1839. Dr. Carpenter then returned to Bristol, where he was appointed Lecturer on Forensic Medicine in the Medical School, and where he commenced the practice of his profession; but in 1843 he came to London with the intention of devoting himself to the pursuit of physiological science. He was soon appointed Lecturer on Physiology at the London Hospital Medical School, and later he became Professor of Medical Jurisprudence at University College, and Examiner in Comparative Anatomy and Physiology in the University of London. In 1856 he was appointed Registrar of the University of London, which office he held with great advantage to the University until the year 1879, when he retired with a pension, and at the earliest vacancy he was appointed by the Crown a member of the Senate, in which capacity he continued to the last to exert a powerful and most beneficial influence.

Dr. Carpenter was the author of numerous well-known and highly popular works. While in Edinburgh he contributed several papers to the medical and scientific journals, and in 1839 was published his prize graduation thesis 'On Physiological Inferences from the Structure of the Nervous System in Invertebrated Animals.' In the same year appeared his earliest systematic work on 'The Principles of General and Comparative Physiology,' a new edition of which was called for in 1841. In later editions the subject was divided, and in 1842 'The Principles of Human Physiology' was published as a separate work, the 'Principles of Comparative Physiology,' henceforth appearing also as a distinct work. Both these works and the 'Manual of Physiology,' which was first published

in 1846, passed through several editions, as did also the well-known and popular work on 'The Microscope and its Revelations,' which first appeared in 1856. The last, and one of the most important of Dr. Carpenter's physiological works, was that entitled 'Principles of Mental Physiology,' 1874. In this work he discusses in the spirit of a true philosopher the strange and perplexing subjects of so-called "mesmerism," "table-turning," "thought-reading," and other phenomena of what is commonly known as "spiritualism." With a large amount of success he laboured to separate the authentic facts from the results of fraud and imposture, and while he denounced the latter he showed that the former, incredible as they may at first sight appear, admit of a strictly physiological explanation.

Dr. Carpenter contributed to the 'Philosophical Transactions' several papers on the "Foraminifera" and other subjects. He also took an active part in promoting the expeditions for deep-sea exploration, for which purpose the "Challenger" was despatched. His reports of these expeditions are contained in the 'Proceedings of the Royal Society' and the 'Journal of the Royal Geographical Society.' For several years Dr. Carpenter was the editor of the 'Medico-Chirurgical Review.' He was elected a Fellow of the Royal Society in 1844, and in 1861 the Society voted him a Royal Medal for his physiological researches. The University of Edinburgh conferred on him the honorary degree of LL.D. in 1871. In 1872 he was President of the British Association at its Brighton Meeting. In 1873 he was elected a corresponding member of the Institute of France, and in 1875 he was created a C.B. in recognition of his services to the University of London. In 1883 he was elected an Honorary Fellow of this Society.

Dr. Carpenter's last published writing was a letter which appeared in the 'Times,' in which he contended against the arguments employed by certain opponents of vaccination. Few men were so well qualified as he was

to expose the fallacious statements of anti-vaccination fanatics.

Dr. Carpenter's death, which occurred on the 10th of November last, was the result of accidental burns occasioned by the overturning of the lamp of a hot-air bath. It scarcely need be added that Dr. Carpenter was universally held in the highest esteem, not only for the extent and variety of his scientific attainments, but also on account of his high principles and his stainless life.

Professor Frederick Gustavus Jacob Henle[1] was born at Fürth in Bavaria, in 1809. When twenty-one years of age he became a pupil of Rudolphi and afterwards of Johannes Müller. When Müller was appointed Professor in the University of Berlin Henle became his Prosector, and taught not only anatomy and physiology, but also pathological anatomy and pathology. In 1840 Henle was appointed Professor of Anatomy at Zürich, and four years later he obtained the Chair of Anatomy and Physiology at Heidelberg, where again he taught pathology in addition to anatomy and physiology. Once more, in 1852, he migrated from Heidelberg to Göttingen, where he continued to work for the remaining thirty-three years of his long and laborious life. He died on the 13th of May last in the seventy-sixth year of his age. He was elected a Foreign Honorary Fellow of this Society in 1859. The name of Henle, and his great reputation as an Anatomist, Physiologist, and Pathologist must be familiar, not only to every anatomist but to almost every practitioner of medicine throughout the civilised world.

In addition to numerous important separate papers and reports, including his annual reports of the progress of anatomy and physiology in the 'Zeitschrift für rationelle Medicin,' Henle was the author of several works of great value. Of these the first in the order of publication was his 'General Anatomy' ('Allgemeine Anatomie'), 1841. Next the 'Handbook of Rational Pathology' ('Handbuch der rationellen Pathologie'), 2 vols., 1846—53.

[1] 'Proceedings of the Royal Society,' No. 239.

Then the 'Handbook of Systematic or Descriptive Anatomy' ('Handbuch der systematischen Anatomie des Menschen'), 3 vols., 1855—71. In 1862 appeared his 'Monograph on the Anatomy of the Kidney' ('Zur Anatomie der Niere'). In this treatise the author described the looped tubes which have been named after him, and which he supposed to be connected with the Malpighian bodies, but to have no openings into the pelvis of the kidney, while he concluded the urine-secreting open tubes to be unconnected with the Malpighian bodies. Most competent observers who have investigated this question are agreed that Henle's conclusions were erroneous[1] and that he greatly exaggerated the number of the looped tubes in the cones of the kidney.

One of the most interesting and important of Henle's anatomical discoveries was that of the muscularity of the middle coat of the arterioles, which he clearly described and figured in his 'Allgemeine Anatomie' in 1841 (p. 498, Plate III, figs. 8, 9, and 10). This discovery formed the anatomical basis for the experiments and conclusions of Brown-Séquard and Bernard which led to our present knowledge of the regulating function of the muscular arterioles and of the vaso-motor nerves. And assuredly until this knowledge had been acquired we were but imperfectly acquainted with the forces which are concerned in effecting and regulating the circulation of the blood. It has now been proved to demonstration that the muscular force possessed by these Lilliputian canals is so great that the united forcible contraction of the pulmonary or of the systemic arterioles is more than equal to the propulsive power of the corresponding right or left ventricle of the heart, and in consequence the onward movement of the blood may be thereby arrested.

This arrest of the circulation by the contraction of the muscular arterioles is most easily demonstrated in the lungs. When, from any cause, the aeration of the blood

[1] See Dr. Beale on 'Kidney Diseases, &c.,' 1869, p. 10.

is prevented, the animal dies in a few minutes and the chest being opened immediately after death, the right cavities of the heart are found to be enormously distended, while those on the left side are nearly empty. The immediate cause of death has been the arrest of the blood by the forcible contraction of the pulmonary arterioles.

Physiologists all agree in teaching that the function of the arterioles is to regulate the blood-supply to the tissues, —to exert, in short, what I have ventured to call a "stop-cock" action upon the blood stream. But there is not the same agreement amongst pathologists.[1] Thus the learned and eloquent Bradshawe Lecturer at the Royal College of Physicians, last August,[2] maintained, in opposition, as he admitted, to the teaching of modern physiologists, that the now generally recognised hypertrophy of the muscular arterioles in cases of chronic Bright's disease is the result, not of over-action in opposition to the heart, but of an "effort of the entire muscular element of the circulatory system to forward a fluid to which the absorptive or appropriative powers of the tissues are ill adapted." It is unnecessary to say that if this doctrine of the propelling power of the muscular arterioles is true the physiologists are all wrong. And in reply to Dr. Goodhart's objection to the "stop-cock" theory, that there is no such antagonism in nature as that would imply, I need only refer to the notorious fact that muscular antagonism, in the case of both voluntary and involuntary muscles, with resulting physiological harmony is of constant occurrence. Amongst voluntary muscles there is the orderly antagonism of flexors and extensors, abductors and adductors, pronators and supinators. In the case of muscles only partly voluntary, those of inspiration and expiration, the sphincters and detrusor muscles are opposed, while amongst

[1] So little acquainted are some controversialists with the physiology of the circulation that they refer to the doctrine of contraction of the arterioles as a regulating influence, as if it were a theory of my own, and they actually compare it with Cullen's hypothesis of spasm of the extreme vessels!

[2] 'Lancet,' August 22nd, 1885.

purely involuntary muscles the radiating and circular fibres of the iris, though directly antagonistic, work together with perfect harmony. And so, it is probable, do the propelling heart and the regulating muscular arterioles co-operate in carrying on the circulation of the blood both in health and in disease.

A consideration of the many important physiological and pathological phenomena which depend for their solution upon a knowledge of the structure and function of the muscular arterioles suffices to show that Henle, by this single anatomical discovery, conferred a great benefit upon mankind. In his doctrine of the etiology of contagious diseases, Henle anticipated in a general way the more exact discoveries of later years. He maintained that the material of contagium is not only organic, but organised and living, and that it must consist of "parasitical beings which are among the lowliest and smallest, but the most productive which are known."

Dr. Noël Gueneau de Mussy[1] was a highly distinguished and accomplished French physician, whose death in Paris, at the age of seventy-two, after a long and painful illness, occurred in May last. After a brilliant student career he became Chomel's Chef de Clinique in 1839, Physician to the Hôtel Dieu in 1842, Assistant Professor of the Faculty of Medicine in 1847, and Member of the Academy of Medicine in 1867. This Society elected him a Foreign Honorary Fellow in 1878.

He is said to have been a highly successful clinical teacher, while the dignity of his character, the extreme affability of his manner, and his scientific ability rendered him, for a number of years, one of the leading physicians of Paris. He was connected with England by the tie of marriage, and he was a frequent attendant at the meetings of the British Medical Association and a valued contributor, on French topics, to the 'British Medical Journal.'

The subject of this notice was the cousin of Dr. Henri

[1] 'Medical Times and Gazette,' June 13th, 1885; 'British Medical Journal, June 6th, 1885.

Gueneau de Mussy, who, after the French revolution in 1848, came with the exiled Orleans family to London, where he was a highly esteemed and successful physician, until, after the deposition of the late Emperor Napoleon, he again returned to Paris.

Professor Henri Milne Edwards[1] was born at Bruges in October 1800. Having completed his elementary studies in Belgium he studied medicine in Paris, where he graduated in 1823. While continuing through life to take an interest in medical subjects he soon gave up the practice of his profession and devoted himself to the study of natural history, and especially to researches among the lower forms of animal life.

During the years 1826 and 1828, in company with his friend and fellow-labourer, Audouin, he made a careful study of the various vertebrates on the coasts of Granville, around the isles of Chaussey, and as far as Cape Frehel. A member of the French Academy was at that time engaged on some hydrographical work off this coast, and he assisted the two naturalists by enabling them to use the dredge in deeper water than they could reach from a row-boat. The results of these investigations were laid before the Academy of Sciences in 1829 and formed the subject of an elaborate laudatory report by Baron Cuvier, which was presented to the Academy in November, 1830. The researches thus commenced were continued by Milne-Edwards throughout his long life.

In 1841 he was appointed Professor of Natural History in the Collége Royal de Henri IV, and about the same time he held the Chair of Zoology and Comparative Physiology at the Faculty of Sciences, of which Faculty he was afterwards the Dean. On his friend Audouin's death he became Professor of Entomology at the Museum of the Jardin des Plantes. About this time he published numerous original memoirs in the 'Annales des Sciences Naturelles,' of which famous periodical Milne-Edwards was for fifty years one of the editors.

[1] 'Nature,' Aug. 6th, 1885.

In addition to his reputation for original research he became widely known and popular by the publication of his elementary works on zoology. His 'Éléments de Zoologie,' published in 1834, was reissued in 1851 under the title of 'Cours Élémentaire de Zoologie.' This work had a very large circulation and was translated into several languages.

Amongst his more important separate works may be mentioned his 'Histoire Naturelle des Crustacés,' 1834-40, in which he was assisted by his friend Audouin; the 'Histoire Naturelle des Coralliaires,' 1857-60, with which was associated another friend, Jules Haime. The 'Leçons sur la Physiologie et l'Anatomie comparée de l'Homme et des Animaux,' published between 1857 and 1882 in fourteen volumes, were dedicated to his friend, M. J. Dumas. 'Recherches Anatomiques et Physiologiques pendant un Voyage sur les Côtes de la Sicile, &c.,' forms a quarto volume of more than 850 pages, illustrated by nearly 100 coloured plates.

For a number of years Milne Edwards was one of the leaders of zoological science. He was one of the first naturalists who made prolonged visits to the sea coast to study the living forms of animal life and to investigate their habits. His investigation of the lower forms of invertebrate animals led him to the theory of there being distinct centres of creation, and this theory is said to have prevented his full and complete acceptance of Darwin's wider generalisation.

In 1838 he was elected a Member of the Academy of Sciences, in the section of Anatomy and Zoology. He was made an Officer of the Legion of Honour in 1847, and a Commander of the Order in 1861. In 1862 he succeeded Geoffroy Saint-Hilaire as Professor of Zoology at the Jardin des Plantes, and soon afterwards he became Assistant Director of the Museum. He was elected an Honorary Fellow of this Society in 1876, and he was a member of most of the learned societies of Europe and America. He died in Paris on the 29th of July last.

If, now, for a moment, we contemplate the work accomplished by the twenty-one men who have recently been taken from our midst, who shall estimate its value? While some —a minority it must be confessed—with a genius for discovery, were enabled to extend the boundaries of our knowledge, and so to confer untold benefits upon all future ages of mankind, there is not one amongst them who has not, in proportion to his ability and his opportunity, been a public benefactor, and as such has earned the gratitude of his contemporaries. Now we trust "that they may rest from their labours, and their works do follow them."

It will be in the recollection of the Society that in my address last year I referred to the subject of the lighting and ventilation of this room as one which would demand the attention of the Council. Without loss of time the Council appointed a sub-committee to inquire and report upon this important matter. And, in the first instance, the question of lighting by electricity was carefully considered. We felt that if the products of gas combustion could be got rid of we should secure the double advantage of a more wholesome atmosphere throughout the building, and a diminished annual expenditure for bookbinding. We therefore obtained from two firms an estimate of the primary cost and the annual expenditure that would be incurred if lighting by electricity were adopted. The estimates given by the two firms were almost identical, and they were to this effect:—The immediate outlay for machinery and fittings would be about £500, and the annual cost of gas for the engine would be somewhat in excess of that which is entailed by our present consump tion of gas.

Then, in reply to our inquiry, it was admitted that the vibration and noise caused by the gas engine, which would have to be placed in the basement immediately beneath the floor of this room, might be a source of annoyance during our meetings. Therefore, after due consideration, the Council unanimously decided not to incur the large expenditure and the probable annoyance which the scheme

of electric lighting would at present involve. And they had the less difficulty in arriving at this decision from the consideration that probably at no very distant period the means of electric lighting will be supplied by public companies at a comparatively small cost and without the noise and vibration attending the generation of electricity by an engine working on our own premises. I have no doubt that this decision of the Council will be confirmed and approved by the Society.

Meanwhile we had to consider the best means of improving the lighting and ventilation of this room. The outside metal tube which conveys the products of combustion from the sun-light had become corroded and had broken off. It was necessary that this should be renewed, and in doing this the opportunity was taken to increase the number of burners and at the same time to improve the ventilation by giving additional facility for the escape of the heated air.

The increased illumination which has thus been obtained from the sun-light enables those who sit at this table to dispense with the two large gas burners which have always hitherto been in use, and as a result the heating and contamination of the air have been very materially lessened.

In the adjoining back room the illumination has been much improved. Some years since two sun-lights were fixed immediately beneath the ceiling, in fact so close to the ceiling as to expose the floor above to the risk of ignition. This danger was felt to be so great that from the first the use of those sun-lights was forbidden. Now the burners have been brought down to a distance of about twelve feet from the ceiling, and the products of combustion are effectually carried off by trumpet-shaped tubes suspended above them. By this change, while improved ventilation and increased illumination have been obtained, the risk of overheating the ceiling and floor above has been entirely removed.

It will be observed that the expense of these alterations following upon the large expenditure involved in the im-

portant drainage works last year leaves us in debt to our bankers; but as the receipts of the annual subscriptions will restore the balance in a few weeks, and as no such extraordinary expenditure is likely to be called for in future, the Council have deemed it undesirable to sell out stock, the annual income of the Society being about £200 in excess of the ordinary expenditure.

The discussion on cholera, which in my last year's address I announced that I had undertaken to initiate, occupied two evenings during the month of March, and brought together a large number of Fellows and Visitors, many of whom took part in the debate.

The discussion, if it did not materially increase our knowledge of the subject, served to bring into view the very contradictory opinions which are held not only with regard to the etiology, the infectiousness, the pathology, and the treatment of the disease, but also with reference to such easily demonstrable and often demonstrated anatomical facts as the relative amount of blood on the two sides of the heart when the chest is opened soon after death during the stage of collapse.[1]

Amongst the subjects which excited most interest and which were most fully discussed was that of Dr. Koch's comma-bacillus and its relation to the disease. Upon that question I did not then venture to express any opinion, but Dr. Koch's later observations and experiments, as related by him in his speech at the opening of the Cholera Congress at Berlin in May last,[2] many of which have been repeated and confirmed by Mr. Watson Cheyne[3] and other competent and trustworthy observers, appear to render it at least highly probable that the comma-bacillus is not only constantly associated with Asiatic cholera, but that it is the morbific agent by which the disease is propagated.

[1] See the report of the discussion, 'Proceedings of the Royal Med. and Chir. Soc.,' new series, vol. i, pp. 392—420.

[2] 'British Medical Journal,' Jan. 2nd and 9th, 1886.

[3] "Reports to the Scientific Grants Committee of the British Medical Association," 'British Medical Journal,' April 25th *et seq.*, 1885.

After a series of carefully conducted experiments Dr. Koch discovered a certain method of inducing cholera in guinea-pigs by introducing the bacilli into the stomach of the animal. And one of the most interesting and practically instructive facts which he records is that, in order to ensure the deadly action of the infecting material, it is necessary to prevent its too rapid escape from the intestinal canal by the narcotic effect of opium injected into the cavity of the peritoneum, the object being to arrest or retard peristaltic movement, and so to render it possible, as he says, "for the comma-bacilli to remain longer and gain a footing in the intestine." The result of this experiment of Koch's is quite in accordance with my own observation that the abrupt arrest of choleraic diarrhœa by opium prevents or retards the escape of the poison, and is often followed by fatal collapse. Additional evidence of the pathogenic power of the cholera bacilli is afforded by the case of a physician who got a severe attack of cholera at a time when the only possible source of infection was the incautious manipulation of the cholera bacilli in Dr. Koch's laboratory. The intestinal discharges in that case contained very numerous cholera bacilli.

It will be seen from the report of the Council that the attendance of Fellows and visitors at the meetings and the number of those who have taken part in the discussions during the past year have been above the average, while the last volume of our 'Transactions' will bear comparison with its predecessors for the interest and importance of the papers which it contains. The Council, too, have received a large number of interesting papers for future reading and discussion.

The publication of the discussions on the papers which are read before the Society in the 'Proceedings,' a practice which was initiated during my predecessor's tenure of office, has proved a complete success, and has added greatly to the value and interest of the 'Proceedings.'

In now retiring from the Presidential Chair, which by

your favour I have been privileged to occupy during the past two years, I do so with a very grateful sense of the honour which has thus been conferred upon me, and with a most fervent and heartfelt wish for the continued prosperity and usefulness of this the greatest of the medical societies in the United Kingdom.

DIFFUSE LIPOMA.

BY

W. MORRANT BAKER, F.R.C.S.,

SURGEON TO ST. BARTHOLOMEW'S HOSPITAL; CONSULTING SURGEON TO THE EVELINA HOSPITAL FOR SICK CHILDREN.

AND

ANTHONY A. BOWLBY, F.R.C.S.,

SURGICAL REGISTRAR AND DEMONSTRATOR OF SURGICAL MORBID ANATOMY AT ST. BARTHOLOMEW'S HOSPITAL.

Received March 10th—Read October 27th, 1885.

The term diffuse lipoma is applied by the authors to certain cases in which there is a great local increase of the subcutaneous fat, without any distinct boundary or capsule such as is usual in the more common forms of circumscribed lipomata.

These growths are generally symmetrical, and are most common over the mastoid processes, in the nape of the neck, and in the submaxillary regions. As will be seen by reference to the cases about to be described they are, however, met with in other situations.

In the 'Transactions of the Pathological Society of London,' vol. xxx, 1879, p. 417, a case is recorded by one of the writers (Mr. Morrant Baker) in which the patient was the subject of these tumours which occupied the upper and back part of the neck and the submaxillary regions.

The tumours are thus described: "There are two tumours in the scalp, symmetrically situated, one on and behind each mastoid process; two, also symmetrical, in the neck—one in each anterior triangle—and a fifth at the back of the neck, in the median line. When the patient was first seen, about two months ago, the tumours over the mastoid processes were of about the size of a small Tangerine orange; those in the anterior triangle, less defined at their margins, occupied the upper two thirds of this space; and the post-cervical tumour, of a circular outline, and less prominent than either of the others, had a diameter of about three inches.

"All the tumours have the same general characters. They are soft, almost fluctuating, yet not tense; apparently seated in the subcutaneous tissue, yet not easily movable on subjacent parts. The skin over them, with which they seem continuous, is not altered in colour or texture, and they are not in the least degree tender. Their consistence seems to lie somewhere between that of a soft fatty tumour and a subcutaneous nævus; the tumours cannot, however, be obliterated so completely by pressure as the latter, nor are they lobulated like the former. Their texture feels most like that of the soft, semi-fluctuating, fatty lumps in the lower part of the neck which accompany the condition known as sporadic cretinism."

In a postscript, dated May 24th, 1879, it is noted that "the tumours have suddenly undergone a great alteration. The post-mastoid tumours now feel almost as tense as if they were distended by fluid, and the post-cervical median tumour, from a state in which it was scarcely distinguishable from the surrounding subcutaneous tissue, is now in all respects indistinguishable from an ordinary well-defined, firm and lobulated, fatty tumour. Were it the only tumour present, and were the history unknown, no surgeon could hesitate for a moment in diagnosing it as an ordinary fatty tumour."

From the great variations in size to which these

tumours were subject in this particular case, from their want of definition, and from the condition of the general health of the patient, it seemed probable that they were rather lymphadenomatous than fatty. A short time before the patient's death, which occurred from some obscure disease of the lungs, a year or two after his exhibition to the Pathological Society, the tumours were said to have almost completely disappeared. It seemed probable at the time that there might be some pathological connection between the tumours in the neck and the persistent bad health of the patient, which ended in death; but examination of many subsequent cases of similar tumours, without accompanying general disease, suggests that in the first recorded case the connection may have been only accidental.

The description of the tumours quoted above is applicable to all the cases now to be recorded, but in no subsequent individual case have the variations in size and consistence been so noticeable. In no case, moreover, has the affection of the general health been so marked, with the exception of Case No. 6, in which the patient was the subject of advanced phthisis. In most of the cases, too, the tumours have been larger and more tense, and in some of the post-mastoid tumours the bases have been so hard as to suggest the presence of much fibrous tissue.

A glance at one of the drawings will give a much better idea of the general aspect of a case than any verbal description. (See Plates I and II.)

Although the disease is seen in its most typical form in the neck and occiput it is not confined to these regions. The tendency to the growth of adipose tissue is in many of the cases observable in the arms and forearms, the scrotum, abdominal wall, and the inguinal and hypogastric regions. In one case large masses of fat were found in the mammary regions (Plate II, fig. 2). This is the only case in the series in which the post-mastoid tumours were so small as to be scarcely noticeable.

The total number of cases observed by us is thirteen.

Case 1.—N. D— (Jan. 25th, 1883), a strong, healthy looking man, æt. 45, says he has always enjoyed good health. He is employed as an ostler; has no visceral disease, but owns to drinking a great deal of beer, and some gin, and other spirits. He does not get drunk, but is often tippling; occasionally vomits in the morning, and more often simply retches. His tongue is tremulous, raw, and inflamed, a typical drunkard's tongue.

At the back of the neck and extending over each mastoid process are symmetrically-placed swellings, limited above by a line prolonged backwards from the zygoma, and below less distinctly limited. Their upper portion is firm, fixed, and resistant, and their outline smooth and rounded; below they are softer, and more inclined to be lobulated. The swelling is largest on the right side, and measures $5\frac{1}{4}$ inches in its transverse diameter, by $3\frac{1}{2}$ inches from above downwards. On the opposite side the measurements are respectively 5 inches and 3 inches. The submaxillary region is occupied by a soft pendulous mass, largest under the right side of the lower jaw, looking like a double chin (Plate I, fig. 2). Its consistence is irregular, and in some places hard masses like enlarged glands can be felt. The right groin presents a small swelling over the femoral glands; the left groin one about twice the size of its fellow; the glands themselves cannot be distinctly felt.

There are no tumours in any other parts of the body. Some of these masses have been noticed by the patient for about twelve months, but those in the groin had not attracted his attention. He says the swellings increase in size, but vary at different times.

Urine and blood normal.

March 8th.—The patient has been under Dr. Andrew, of Hendon, and has taken Liquor Potassæ without much change in the swellings. They are perhaps a little softer.

Case 2.—J. C—, æt. 40, is in good health, works hard at a wine and spirit merchant's, mostly as a warehouse-

man, and says he can easily carry two hundredweight on his back. No visceral disease; says he drinks a great deal of gin. Urine normal. On the back of the neck, over the upper cervical vertebræ, is a large swelling occupying each side of the sub-occipital region, extending equally over the mastoid processes, and having a marked median groove along the line of the spine; the appearance indicates that the tumour commenced in two lateral growths, which subsequently met across the middle line. The upper limit of the swelling on each side is about on a level with the tip of the ear. The diameter transversely is $7\frac{1}{2}$ inches; from above downwards $4\frac{1}{2}$ inches.

The whole of the submaxillary region is occupied by a large, semi-fluctuating mass, which extends upward over each cheek, and presents no median division. Its measurement from one cheek to the other is 12 inches; the upper boundary is harder to the touch than is the lower part of the swelling; the skin over it is slightly red.

Masses similar to the above are found on the upper arms, more especially on the left, the circumference of which is 16 inches, that of the right being $14\frac{1}{2}$; the supraclavicular regions are free.

In both groins, particularly the right, it seems as if the glands were embedded in swellings, which feel as if composed of tissue similar to that forming the growths in other parts. On the outer side of each thigh are tumours of a similar nature, though small; and below the umbilicus there is a collection of a like material.

The patient can give no very definite history, but says that the various lumps began to grow about four years ago. He thinks that some of them, especially those on the neck, are still increasing.

Case 3.—J. M—, æt. 51, is in good health. Thoracic viscera normal. A little pale and pinched about the face, but has a good deal of subcutaneous fat about the body.

Urine acid, and contains a trace of albumen. Says he drinks a great deal of gin. Digestion bad.

In the centre of the back of the neck is a large tumour of a rounded shape (Plate I, fig. 1). It extends about an equal distance on each side of the middle line, the situation of which is marked by a barely perceptible groove. The transverse diameter measures 5 inches, and the thickness of the tumour is about 3 inches.

Higher up the neck on each side, behind the ears and over the mastoid process, are two swellings of a similar kind. That on the left side is the larger, and is about 5 inches in diameter; its outline is nearly circular, and the skin over it is red, and rather tender, though not in any way indicative of impending suppuration.

The tumour on the opposite side is about 3 inches in diameter, also of a rounded shape, and covered by normal skin. Neither tumour encroaches on the middle line of the neck.

The patient says that the large tumour has been growing for seven years, the smaller ones four or five years. In his opinion they are at some times smaller than at others. No similar swellings exist in other parts of the body.

Case 4.—D. L—, æt. 38, car-driver, has suffered from chronic bronchitis for about four years, but is otherwise healthy. Drinks a great deal of beer and spirits. Appetite bad. Pain in loins. Urine acid; contains a good deal of albumen.

Symmetrically placed on each side of the upper part of the neck, and over the posterior portion of each mastoid process, are two lumps—each about twice the size of a small hen's egg—slightly crossing the middle line, along which is a deep longitudinal groove. The upper boundary of each lump is a line drawn backwards from the zygoma. Their measurements are 4 inches long by 3 wide.

Under the skin in the submaxillary region is a soft diffuse swelling, not extending into the cheeks. A small

swelling about the size of a walnut is placed on each zygomatic arch immediately in front of each ear, that on the right side being rather the larger. Lumps of similar size are found on each side of the spine in the lumbar region.

In each groin the glands appear hidden and involved in similar growths. The scrotum is enlarged by the presence of similar soft growths, and is pendulous. Both arms and forearms are very much enlarged and misshapen by diffuse soft masses in the subcutaneous tissue, feeling like fat. In the left arm the lumps are much more circumscribed below the elbow. The greatest circumferences of the arms and forearms are as follows:

R. arm . 12¼ inches; R. forearm . 11⅛ inches.
L. arm . 14 inches; L. forearm . 12⅛ inches.

The history the patient gives is that the mass on the right side of the nape of the neck began to grow three years ago, and was soon followed by the appearance of its fellow; the submaxillary region, groins, and arms were then affected in order, the swellings in the latter being noticed eighteen months ago. He is not sure that the tumours are still growing, and says that they vary in size. This latter statement is certainly correct, for a week after the above description was written the tumours in the neck were distinctly smaller and less tense.

Case 5.—J. C—, æt. 48, has been a healthy man, but owns to having drunk much spirits, chiefly rum, often as much as eight glasses a day; has not drunk so much lately. No appetite for food; suffers from nausea.

For two years he has noticed lumps on his neck, which have become much larger during the last six months, and which he thinks are still growing. He thinks they vary in size; they cause no pain.

On the back of the neck on each side are two large masses very nearly equal in size—that on the right being rather the larger—and partly subdivided by a transverse groove. Their greatest diameters are in the long axis of

the body, and measure $4\frac{1}{2}$ inches each; transverse diameter of the right $2\frac{3}{4}$ inches, of the left $2\frac{1}{2}$ inches. In front of each ear is a small swelling on the zygoma, that on the left side being the larger, and about as big as half a walnut.

Has no swellings in other parts of the body.

February 9th.—Has been taking Liq. Potassæ for the past month, with the result that the swelling over the right mastoid process is smaller and softer. No other change.

March 12th.—Has continued Liq. Potassæ. No improvement.

Case 6.—C. S—, pig-slaughterer, æt. 33. Married, and has two children, aged five and four years. Says he has been a fairly healthy man, but has lately been troubled with cough. Has drunk much, chiefly beer and spirits. Hand tremulous; tongue glazed and superficially ulcerated. Phthisis at right apex. Liver enlarged. Urine acid, loaded with blood, which has been present for the past week. Pain in the loins. Fistula in ano of five months' duration.

On the upper part of the back of the neck are two symmetrically placed swellings, each $4\frac{1}{2}$ inches long by about $2\frac{3}{4}$ inches wide, limited above by a line prolonged backwards from the zygoma, and each partially subdivided into two equal portions by a transverse groove, which is most marked on the right side; the portion of the tumour above the groove is firmer and more elastic than that below, which is softer and less defined. The left submaxillary region is occupied by a large, soft, pendulous mass, ill-defined in all directions, the right side of the neck being but slightly affected. The lymphatic glands in each groin are hidden by soft tumour-like masses of an apparently similar nature to those in the rest of the body, but of small size. There is a slight swelling on each side just above the pubes, about the size of a marble.

The patient thinks the lumps have been growing for about two years, but is not certain. He thinks they vary in size.

Case 7.—W. H— (Nov., 1883), a healthy man, æt. 29, of healthy parents. Drinks about six quarts of beer daily, three quarts of milk, and half a pint of gin. Eats little meat, and is fond of sucking raw eggs to the amount of five or six a day. No visceral disease. Digestion and general health good. Has noticed swellings on the breast, abdomen, and in the groins for twelve months. They all appeared simultaneously and are increasing. They do not vary in size. A lump on the left side of the neck appeared at the same time as the others.

The pectoral regions are occupied by large globular swellings, leading one to suppose at first sight that the patient has unusually developed mammary glands. They are of equal size, each about as large as the average mamma of an unmarried woman (Plate II, fig. 2).

Over the middle line of the abdomen are large rounded swellings, limited laterally by the lineæ semilunares, and transversely constricted by the lineæ transversæ. There is a soft mass over the pubic bone. The glands in each groin and in the right axilla are embedded in soft swellings.

The upper and inner part of each arm is occupied by a soft pendulous outgrowth, the whole limb being in each case much enlarged, so that the greatest circumference of the right arm is $14\frac{3}{4}$ inches, that of the left $14\frac{1}{8}$ inches.

In the left submaxillary region is a swelling as large as an egg, irregular in outline, and pendulous. From the hyoid bone to the lobule of the left ear the measurement is $6\frac{1}{4}$ inches, a similar measurement on the right side being 5 inches. There are two symmetrical swellings in the scrotum, one behind each testis.

All the tumours have a soft doughy feel, and are evidently composed of fat. The skin over them is mostly

adherent, especially over those on the arms, and dimples when pinched up. There are symmetrically-placed swellings behind the mastoid processes, but of small size, and hardly noticeable.

February 16th, 1884.—All the swellings have greatly increased. In each pectoral region is a large rounded mass, as big as a full-sized female breast, and with the nipple in its centre. General health good. Says he has given up spirits, but drinks beer.

Case 8.—F. B—, æt. 41 (September 29th, 1883), a weak, unhealthy-looking man. Is said to be of temperate habits. For two years has noticed swellings in his neck, and says that for the last year they have been very painful. In the middle line of the neck in the submaxillary region is a large, soft, pendulous swelling. Behind each mastoid process is a rounded swelling, extending from the superior curved line of the occipital bone to the sixth cervical vertebra. These swellings are united across the middle line in the lower half of their extent. Extending along the middle line, and on each side of it, from the first to the fourth dorsal vertebra, is a similar mass of soft tissue feeling like fat. At their upper boundary the tumours are of firm consistence.

The patient was treated with Liq. Arsen., but did not improve.

Case 9.—W. P—, æt. 38, hairdresser, admitted into St. Bartholomew's Hospital under the care of Mr. Willett, March 2nd, 1885. A wasted, unhealthy-looking man. Family history of phthisis. Winter cough for some years past. No material pulmonary disease. Urine normal. Has lateral curvature of the spine.

For many years he has been in the habit of drinking large quantities of spirits, often as much as half a pint to a pint of brandy daily. He also drinks beer. The spirits are consumed at frequent intervals in small quantities, and he says he is never intoxicated.

Behind each mastoid process is a swelling the size of

half an egg, rounded and smooth to the touch, firm above, where it is limited by the superior curved line of the occipital bone, but more soft and less well defined at its lower border.

In each parotid region, immediately in front of the ear, is a small rounded swelling as big as a walnut, soft, painless, and compressible.

Symmetrical swellings of similar size to those in the parotid region are found in the upper part of the scrotum. They are freely movable.

In the perinæum is an irregular and very ill-defined soft mass, extending from the scrotum to the anus, symmetrically distributed on each side of the middle line, and with its long axis in an antero-posterior direction. It is distinctly lobulated, and though movable on the deeper structures is in parts adherent to the skin.

Over each external abdominal ring is a rounded softish swelling about an inch in diameter, the skin over which is partly adherent.

In the abdominal wall, on each side of the middle line, below the umbilicus, are symmetrical swellings each as large as half an orange.

The preceding cases, ten in number, including the one already described in the Pathological Society's 'Transactions,' have been observed by us at St. Bartholomew's Hospital. For the following we are indebted to Dr. Allchin, Dr. de Havilland Hall, and Mr. Henry Morris.

Case 11. (From Mr. Henry Morris.)—R. R—, æt. 63, steward on board a steam-packet, is suffering from cancer of the mouth and tongue. For thirty-seven years has noticed the tumours about to be described. Twenty years since Mr. Cock removed two of the smaller ones from the neck; the others continued to grow until ten years ago.

There are now three tumours at the back of the neck, one on the right and two on the left side of the well-marked and easily felt ligamentum nuchæ. There is also an enormous, soft, pendulous, almost diffluent mass, which

extends from below the ear on one side, beneath the chin to the same point below the other ear. It hangs over the top of the chest.

Case 12. (From Dr. de Havilland Hall.)—J. L—, æt. 44, has been a healthy man until the last three years. Since then he has suffered from cough, with much expectoration and occasional hæmoptysis. Has been a heavy drinker, taking large quantities of both beer and spirits, often half a pint to a pint of gin daily. Latterly he has not drunk so much spirits, but still consumes large quantities of beer. Is subject to headaches.

A year ago he noticed swellings in the neck; since then they have increased, but are sometimes smaller than at others.

Present condition.—Symmetrically placed behind the mastoid processes are two firm, rounded swellings, each as large as a Tangerine orange, similar to those already described in the previous cases. In the submaxillary region is a soft pendulous swelling not large enough to be very noticeable. In each groin is a soft, fatty mass, which apparently extends into the femoral canal, as it gives a distinct impulse on coughing.

Dr. Allchin has kindly forwarded the note of the following case.

Case 13.—C. St. Q—, æt. 36, was for several months under my observation at the Westminster Hospital during 1884.

Has been a cavalry soldier, and was for some years in India, where he drank freely, chiefly brandy, rarely the native spirit. He has quite ceased drinking for the last few years. The tumours commenced whilst he was drinking.

Says he had syphilis in 1867, but it appears questionable whether it was an infecting chancre, for he states he had no secondary manifestations; was treated with Fowler's solution and iodide of potassium.

In June, 1875, tumours were first noticed behind the ear. Their appearance was attended with slight pain, and were at first small and hard, as if the bone were growing out. Says his mastoid processes were always prominent. These swellings continued to increase in size, and to become softer. They attained their present dimensions in December, 1879, since which time they have remained stationary.

The next tumour to appear was the one on the cervical spines four years ago. This reached its full development in two years, and has, like the preceding, remained stationary since that time. Patient's attention was drawn to this tumour by the chafing of his collar.

The fulness under his chin has existed eighteen months; it is not increasing.

A year ago the swelling in front of the left ear just below the zygoma was pointed out to the patient, who had not previously been aware of its existence. It is not increasing in size.

A few weeks since (*i. e.* about last May), patient first noticed swelling in the right arm. This is more flabby, and not so circumscribed as the other tumours.

Patient was discharged from the army in October, 1879, on account, as he says, of the tumours in the neck, which were attributed to syphilis.

For some two or three years patient has noticed an impairment of general health, and a failing memory, with muscular weakness and loss of weight. But this may, in part, be attributed to bad circumstances and poor living, and in greater part to the effect of tænia, from which he was found to be suffering whilst in the Westminster Hospital.

No treatment was administered for the tumours, and patient left hospital in no way altered, so far as they were concerned.

In vol. xiii of 'St. Thomas's Hospital Reports' Sir William Mac Cormac has reported four cases similar to

those just described. In one of these he removed a portion of the post-mastoid fatty tumours with ultimate benefit to the patient, who was very pleased with the result. But Sir W. Mac Cormac remarks that the removal of the tumours was very tedious, the hæmorrhage copious, and the wound extensive.

At the meeting of the Pathological Society, March 20th, 1883 ('Brit. Med. Journ,' 1883, i, p. 623), "Mr. Jonathan Hutchinson showed a mass of fatty tissue removed from the back of the neck of a man, who had large masses in that situation quite symmetrically arranged. The patient also had tumours symmetrically placed on both arms, and he appeared to have symmetrical hypertrophy of the parotid glands, or the appearances might be due to small masses lying over the glands. On March 19th he had attempted to remove one of the masses, but had not found any distinct limit to the mass, which appeared to be a hypertrophy of the subcutaneous fat, not at all encapsuled, and not therefore to be removed. The mass consisted of very firm fatty tissue, with firm fibrous meshes."

At the meeting of the Ophthalmological Society, July 3rd, 1884,[1] Mr. Jonathan Hutchinson narrated the history of a patient, a Hindoo gentleman, in whom proptosis, first on one side and subsequently on the other, occurred in conjunction with a puffy condition of the face and submaxillary region. The proptosis appeared to be due to an increase of the orbital fat, but no symmetrical enlargements of the neck or elsewhere were noted. Mr. Hutchinson expressed his opinion that this case was analogous to that shown by Mr. Baker at the Pathological Society.

At the time that Mr. Morrant Baker exhibited his patient to the Pathological Society he was not aware that other cases had been recorded, but his attention has been since called to the fact that Sir Benjamin Brodie has placed on record examples of the same disease. His observations on the subject may be here quoted:—

"There is another kind of fatty tumor which occurs

[1] 'Transactions of the Ophthalmological Society,' vol. iv, p. 36.

occasionally, but which has not been, as far as I know, described by surgical writers. In the cases to which I allude the tumor is not well defined; in fact there is no distinct boundary to it, and you cannot say where the natural adipose-structure ends and the morbid growth begins. I will relate to you the history of one of several cases of this kind that I have met with, and this will explain as much as I know of the matter. A man came to this hospital several years ago having a very grotesque appearance; there being an enormous double chin (as it is called) hanging down nearly to the sternum, and an immense swelling also on the back of his neck, formed by two large masses one behind each ear, as large as an orange, and connected by a smaller mass between them. He said that the enlargement had begun to show itself three or four years before, and had been increasing ever since. They gave him no pain; nevertheless they made him miserable, and in fact had ruined him. The poor fellow was by occupation a gentleman's servant, and having so strange an appearance no one would take him into his service. I gave him half a drachm of the *liquor potassæ* three times a day, and gradually increased the dose to a drachm, dissolved in small beer. When he had taken the medicine for about a month the tumors were sensibly diminished in size. He went on taking the alkali, and the tumors continued to decrease. It was just then that iodine began to have a reputation, much indeed beyond experience has proved it to deserve, for the cure of morbid growths, and I left off the *liquor potassæ*, and prescribed the tincture of iodine instead. The effect of this change of treatment was remarkable. The patient lost flesh, while the tumors increased in size. Of course I omitted the iodine and prescribed the *liquor potassæ* a second time. Altogether he took a very large quantity of the latter medicine, and left the hospital very much improved, with directions that he should continue to take it, with occasional intermissions. I had lost sight of him for some time when it happened that I was requested to visit a

patient in Mortimer Street. I did not observe the servant who opened the door, but as I was leaving the house he stopped me, saying that he wished to thank me for what I had done for him. It was this very patient. He was so much improved in appearance that he was enabled to obtain a situation as footman. There were still some remains of the tumours, but nothing that was very remarkable. I have seen some other cases of the same kind in which the exhibition of very large doses of *liquor potassæ* appeared to be of great service. But I have not had the opportunity of trying it, or of knowing the results in every case; and I am informed that in some cases it has been given to a considerable extent without manifest advantage." (Lectures on Pathology and Surgery, 1846, p. 275.)

Remarks.—All the cases hitherto observed have been males, the ages varying from twenty-nine to sixty-three years; the majority of the patients being between thirty-five and forty-five years of age at the time the tumours commenced to grow. We believe that all these swellings have a similar structure, being composed simply of adipose tissue; for in the cases in which the tumours were submitted to operation by Mr. Hutchinson and Sir William Mac Cormac, the growths removed consisted of fat. And in several of our own cases the diagnosis has been confirmed by the microscopical examination of portions of the growth removed by Dr. Charcot's "emporte pièce histologique."

The development of these tumours is somewhat rapid. Thus, in case No. 7 they had attained a considerable size within twelve months. The rate of growth, however, varies much in individual cases. Another noticeable fact is that in some instances the swelling varies in size from time to time. Of this fact several of the patients were very certain, and in some we were able to verify their statements. Whether the tumours ever entirely disappear in the absence of any wasting disease we cannot certainly affirm.

With regard to the anatomical position of the swellings we have no doubt that they are situated in the subcutaneous cellular tissue, and we cannot agree with Sir W. Mac Cormac that in the neck they are beneath the fascia of the trapezius muscle.

In support of our opinion we would point out firstly, that in Mr. Hutchinson's case the fatty mass is specially mentioned as being found to be subcutaneous at the time of operation; and secondly that the entirely analogous fatty masses in the submaxillary regions, in the forearms, abdominal wall, &c., are evidently entirely independent of fascial attachments in their growth, being essentially diffuse, absolutely unlimited in any direction and occasionally distinctly attached to the skin.

The manner in which the growths in the post-mastoid regions are limited may also be readily explained without reference to the attachments of the fascia of the trapezius. They are limited above by the superior curved line of the occipital bone, because beyond this limit there is no subcutaneous cellular tissue in which the fat can be developed. In a downward direction these growths are not definitely limited, but in the middle line of the neck there is a more or less well-marked depression, simply due to the fact, which is easily demonstrable, that in this situation the skin is closely bound by strong fibrous bands to the subjacent aponeurosis, and that the subcutaneous tissue is very dense and tough. Nevertheless, the growths may certainly pass across the middle line (see, amongst others, Case No. 2), a condition which would be impossible if they were subfascial.

Sir W. Mac Cormac, indeed, in another part of his paper —possibly by an oversight—says, whilst speaking of the operation, "The mass appeared to consist simply of diffuse subcutaneous fat."

Another point to which we would direct attention is the fact that these fatty masses are prone to develop in the regions occupied by lymphatic glands. Thus they are found behind the ear, in front of the pinna, in the sub-

maxillary and inguinal regions; although they are also frequently present in other situations which have no special connection with the lymphatic glands. Whether the latter glands are ever involved in the growth we are not in a position to state with certainty; but we have not felt them to be definitely enlarged.

Beyond the discomfort produced by the deformity, no symptoms specially referable to these fatty tumours have been observed; and the expression of a wish on the part of one or two of the patients to have an operation performed has arisen only from the unsightliness of the disease.

Internal remedies have apparently little or no effect. In one or two cases, however, the administration of arsenic with steel seemed slightly beneficial. In accordance with Brodie's suggestion we have tried the effect of Liq. Potassæ, but have not hitherto found it beneficial in reducing the size of the growths. We have administered the above-mentioned drugs, as well as iodide of potassium and mercury, in several cases for some months.

As the cases accumulated we had hoped to find some definite conditions which might help in determining the nature or cause of the disease. But the only circumstance which seems to give any clue to its cause is (so far as we have been able to observe) that, with one or two possible exceptions, the patients have been hard drinkers.

Of course this may be an accidental concomitant and even if connected with the disease may be only one element in its further development. But, as will be seen in reading the notes of the individual cases, the fact is too marked a feature to be overlooked. In Sir William Mac Cormac's cases no statement is made with regard to sobriety, but we may remark that the two patients whose occupations were recorded, were, the one a butler, the other a waiter.

The value of alcohol as a fat-forming food is too well known to need much emphasis, but is worthy of notice. It, however, affords no explanation of the great tendency

seen in these cases towards the development of fat in certain regions and not in others.

Appended is a table of the cases to which reference has been made.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' New Series, vol. ii, p. 5.)

No.	Initials.	Age.	Occupation.	Site of tumour.	Duration.	Remarks.
1	N. D.	41	Ostler	Post-mastoid, submaxillary region, and groins	12 mos.	(1) Hard drinker.
2	J. C.	40	Warehouseman at a wine and spirit merchant's	Post-mastoid and back of neck, submaxillary region, arms, and groins	4 years	(1) Hard drinker.
3	J. M.	51	—	Middle of neck at back, post-mastoid regions	7 years	(1) Hard drinker. (2) Albuminuria.
4	D. L.	38	Car-driver	Post-mastoid regions and back of neck, submaxillary region, parotid regions, loins, groins, scrotum, arms, and fore-arms	18 mos.	(1) Hard drinker. (2) Albuminuria. (3) Bronchitis.
5	J. C.	48	Cheesemonger	Post-mastoid regions. Over zygoma on each side	2 years	(1) Hard drinker.
6	C. S.	33	Slaughterer	Post-mastoid and submaxillary regions, groins	2 years	(1) Hard drinker. (2) Phthisis. (3) Hæmaturia.
7	W. H.	29	Cowkeeper	Pectoral regions, abdomen, scrotum, groins, arms, forearms, submaxillary region, and small swellings in post-mastoid regions	1 year	(1) Hard drinker. Also drinks large quantities of milk.
8	F. B.	41	Tailor	Post-mastoid and submaxillary regions	2 years	(1) A temperate man.
9	W. P.	38	Hair-dresser	Post-mastoid regions, scrotum, perineum, abdominal wall	5 mos.	Very intemperate.

10	J. R.	30	Carman	Post-mastoid and submaxillary regions, nape, and anterior triangles of neck	9 mos.	Died of some obscure lung disease. Case recorded in 'Path. Soc. Trans.,' 1879
11	R. R. (Mr. Henry Morris's case)	63	Ship's steward	Post-mastoid and submaxillary regions	30 years	Died with epithelioma of the tongue. Tumours were operated on by Mr. Cock.
12	J. L. (Dr. De Havilland Hall's case)	44	—	Post-mastoid, submaxillary regions, and groin	1 year	(1) Hard drinker. (2) Phthisis. ?
13	C. St. G. (Dr. Allchin's case)	36	Soldier	Post-mastoid regions, back of neck in middle line, submaxillary and parotid regions, arm	9 years	Formerly of intemperate habits.
				Sir William Mac Cormac's Cases.		
1	W. G.	42	Butler	Post-mastoid, back of neck, and submaxillary region	16 years	Partially removed.
2	F. E. W.	63	—	Post-mastoid and back of neck	5 years	—
3	W. S.	44	Waiter	Post-mastoid and back of neck, submaxillary region, abdominal wall, arms	?	Post-mastoid tumours removed.
4	—	60	—	Post-mastoid	3 years	—

DESCRIPTION OF PLATES I AND II.

(Diffuse Lipoma, by W. MORRANT BAKER and A. A. BOWLBY.)

PLATE I.

FIG. 1.—J. M—, Case 3, see p. 45.
FIG. 2.—N. D—, Case 1 see p. 44.

PLATE II.

FIG. 1.—C. S—, Case 6, see p. 48.
FIG. 2.—W. H—, Case 7, see p. 49.

Plate I

Fig. 2.

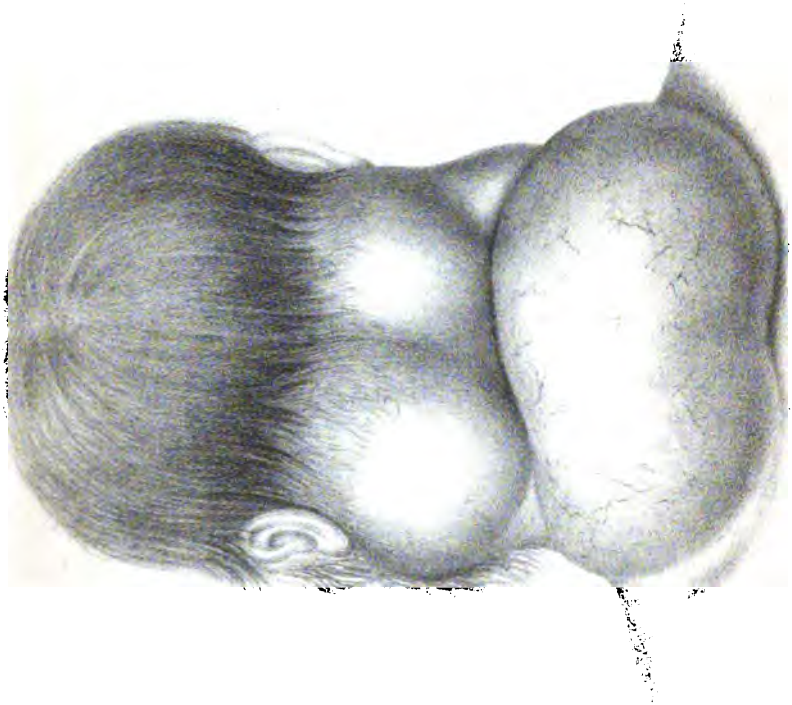

Fig. 1.

Mintern Bros lith

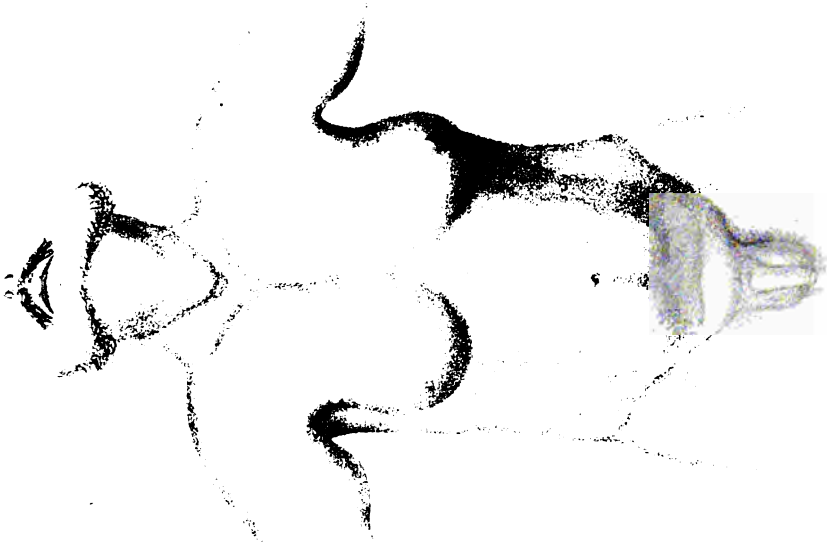

Fig. 2.

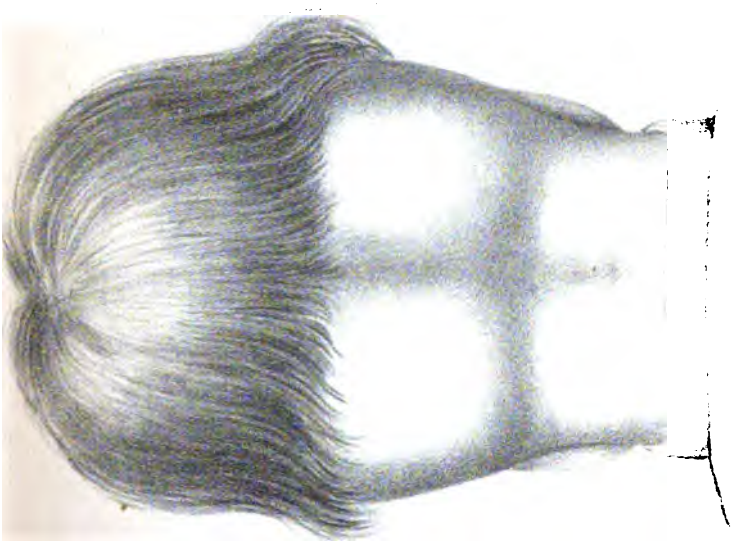

Fig. 1.

Mintern Bros. lith.

A CASE

OF

LIGATURE OF THE LEFT COMMON CAROTID ARTERY

WOUNDED BY A FISH-BONE WHICH HAD PENETRATED THE PHARYNX.

WITH

REMARKS AND AN APPENDIX CONTAINING FORTY-FIVE CASES OF WOUNDS OF BLOOD-VESSELS BY FOREIGN BODIES.

BY

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Penetration of some part of the alimentary canal by sharp-pointed foreign bodies which have been swallowed, and arrested in their passage, is not a very uncommon occurrence. Apart from obstruction to the passage of air to the lungs, or food along the alimentary tract, it is familiar to the surgeon as the cause of two main and distinct kinds of mischief; on the one hand of inflammatory mischief, more or less severe, prolonged, and critical according to the nature of the organ or tissue involved in the imprisonment or migration of the foreign substance, and on the other hand of mischief to adjacent blood-vessels, too often terminating in rapid death from sudden and uncontrollable hæmorrhage. The relative frequency

of the different kinds of fatal lesions due to the arrest of foreign bodies in the pharynx and œsophagus may be gathered from Adelman's table.[1] Out of 314 cases 109 proved fatal, 43 from lesions of the respiratory organs, 25 from ulceration of the œsophagus and inflammatory processes in the neighbouring parts, and 31, or less than one third, from implication of blood-vessels. To the last source of danger attention will mainly be confined in this paper. In the Appendix will be found abstracts of 44 cases in which lesions of blood-vessels occurred through the agency of foreign bodies penetrating the alimentary canal, 43 of these proving fatal. Arranged according to the vessels injured the cases comprise:

23 instances of lesion of the thoracic aorta.
11 instances of lesion of one or more of the carotid arteries.
1 instance of lesion of the left ascending pharyngeal artery.
1 instance of wound of an abnormal right subclavian.
1 instance of wound of the pulmonary artery.
1 instance of lesion of an azygos vein.
1 instance of wound of the heart and right coronary vein.
3 instances of lesion of one or more of the venæ cavæ.
1 certain instance of lesion of the inferior thyroid artery, or one of its branches.
2 doubtful instances of ditto.

45

The different divisions of the alimentary canal enjoy different and unequal liabilities to injury from sharp-pointed foreign bodies.

In the pharynx needles, pins, bristles, and fish-bones readily find a temporary resting-place. Generally they are speedily dislodged and pass along the alimentary

[1] 'Vierteljahrschrift für die praktische Heilkunde,' vol. xcvi, p. 66.

canal; sometimes they continue impacted, and, working their way through the walls of the cavity, either by penetration or ulceration, produce results of a serious or fatal character through inflammatory affections of neighbouring structures, or implication of neighbouring blood-vessels; and these very results may be ensured or aggravated by injudicious procedures adopted for the displacement of the impacted or adherent substances. The part of the pharynx where foreign bodies are most likely to lodge is at its junction with the œsophagus. The pharynx also is liable to be directly perforated, with accompanying wound of one of the carotid arteries by sharp-pointed instruments, or other bodies, such as tobacco pipes thrust through it from the mouth. For some instructive cases of this kind reference may be made to Mr. Durham's able article on "Injuries to the Neck" in Holmes and Hulke's 'System of Surgery,' vol. i, and to the Appendix to this paper.

The narrowness of the œsophagus renders it more especially liable to injury from the lodgment of foreign bodies. The arrest may occur in any part of the tube, the most frequent site being about opposite the point where the left bronchus crosses the aorta. If they are arrested in the neck, the common carotid, and especially the left common carotid, is exposed to danger, and after the carotids one of the œsophageal branches of the inferior thyroid artery. In the thorax the aorta is by far the most frequently injured, but occasionally one of the venæ cavæ, the pulmonary artery, one of the large vessels springing from the arch of the aorta, an azygos vein, or even the heart itself, and one of the coronary arteries or veins may be implicated. Within the abdomen the impaction of foreign bodies is not specially related to lesions of blood-vessels, and I am not acquainted with any cases of wounds of arteries giving rise to fatal hæmorrhage in that cavity. In one case the vena cava was involved. A young woman died in the Middlesex Hospital, after having been ill for fifty-three days, with all

the symptoms of hectic fever, and after having presented the signs of coagulation in the veins of both lower limbs (phlegmasia alba dolens). Throughout the case she complained of aching pains in various regions of the spine. At the autopsy a needle was found in the lower part of the vena cava, and around it a thrombus had formed. There was an opening in the back of the vein about an eighth of an inch in diameter. The iliac and femoral veins on both sides were obstructed. A second needle was found in an abscess to the left of the third lumbar vertebra.[1]

If they reach the rectum, pointed bodies like fish-bones are recognised as occasional causes of ischio-rectal abscess and fistula.

It is a well-known fact that swallowed needles may penetrate the alimentary canal, migrate through the muscles without transfixing any blood-vessel, reach a remote part of the body, and be extracted through the skin. Some remarkable cases of this kind are on record.[2]

[1] Dr. Henry Thompson, 'Brit. Med. Journ.,' 1874, vol. ii, p. 571.

[2] Poulet gives the following:—1. A stepmother, desiring to rid herself of her little daughter, made her swallow at different times a certain number of needles. After a long suffering the needles made their exit from different parts of the body, and especially from the arms. 2. A needle which had been swallowed and lodged in the œsophagus penetrated the muscles, and a month later was found behind the right ear, where it was extracted by an incision. 3. A child had swallowed a needle, which lodged in the œsophagus and pierced its walls; it became embedded in the muscles of the neck. It was extracted by an incision and the aid of a magnet (Kerckringius, 'Spicilegium Anatomicum,' Obs. 44). Lavacherie ('Bull. de l'Acad. Méd. de Belge,' 1848) also mentions the case of a young woman who had a foreign body in the fauces, which, after the lapse of a year, appeared under the skin near the sterno-clavicular articulation, whence it was extracted by an incision three months later.

Poulet adds in a note:—"Vigla has collected the most interesting of these cases of migratory foreign bodies. Hévin quotes several cases in which cornstalks were extracted from abscesses of the thoracic walls thirteen to fifteen days after their ingestion. Bonnet, Helmontius, and Volgnarius have reported similar facts; the latter saw a cornstalk emerge through the axilla. In Polisius' case the stalk made its exit three months afterwards from an abscess in the back. Bally ('Rev. de Méd.,' ii, 1825) reports the ingestion of a stalk; three months later peripneumonia, abscess upon right side of the thorax,

Of the 45 cases of lesions of blood-vessels placed in the appendix 19 resulted from swallowing pieces of bone, 4 were due to sewing needles, 3 to coins, 2 to tobacco pipes, 1 to a puncture by a parasol, 2 to tooth plates, Guthrie's case of wound of both carotids to an ingenious suicidal machine made of corks and pins, whilst 12, including my own case, were caused by fish-bones. In 6 of the 12 the vessel implicated was the thoracic aorta, viz. the cases of Théron, Auvert de Moscou, Bousquet, Dr. Waters, of Liverpool, Dr. Ramskill, and one related in the Catalogue of the Museum of St. Bartholomew's Hospital. I may mention that I witnessed the post-mortem examination on Dr. Ramskill's case, and it was recalled to my mind when I was asked to see the patient whose case forms the basis of this paper. Five of the remaining 6 cases are instances of wound of a carotid, viz. a second case given by Auvert de Moscou, a case briefly referred to by Mr. Cripps in the discussion at the Clinical Society on 24th May, 1878, on Dr. McKeown's paper on a successful case of œsophagotomy for the removal of a set of artificial teeth impacted in the œsophagus, Dr. Reid's case occurring in 1837, my own case, and one under Dr. Cresswell Rich at the Liverpool Royal Infirmary. Some months after the occurrence of my own case, while I was attending the meeting of the British Medical Association at Liverpool in 1883, I saw in the Annual Museum of the Association a specimen showing "perforation of the œsophagus by a fish-bone, with rupture into the left common carotid artery." Through the kindness of Mr. Reginald Harrison, to whom I applied for the particulars, Mr. Paul, and Dr. Cresswell Rich I have been able to append the details of this interesting case, and through the same channel I received the particulars of the case of perforation of the aorta under Dr. Waters above referred to. The sixth case is the well-known case related by Dr. Andrew where a fish-bone penetrated the stomach close to the œsophagus, then the

between the second and third ribs, through which the foreign body emerged." ('Treatise on Foreign Bodies,' vol. i, p. 84.)

diaphragm and pericardium and posterior surface of the heart, and finally inflicted a jagged wound in the middle of the septum immediately over the right coronary artery and vein, penetrating the latter vessel. The pericardium was filled with a pint and a half of fluid blood.

It is worth while remarking that out of the 12 cases of lesions of vessels due or ascribed to fish-bones, the offending bone itself was not certainly discovered in more than 4, viz. Bousquet's, Dr. Andrew's, the case at St. Bartholomew's Hospital, and my own. It was not found in either of the Liverpool cases, the reason doubtless being that it had been washed away by the copious hæmorrhage from the considerable opening at the seat of injury to the artery into a lower part of the alimentary canal. In Dr. Waters's case the opening in the œsophagus was large enough to admit the little finger, and that in the aorta at the junction of the transverse and descending aorta would have admitted a No. 10 catheter, whilst the stomach and duodenum were distended with blood-clot weighing 2¼ lbs., and forming an accurate cast of their cavities. In the case of wound of the carotid, the perforation in the anterior wall of the gullet was circular with perpendicular edge, and of a size to admit a No. 8 catheter, and the opening in the artery was of the same size as that in the gullet. The large bowel was full of altered blood. Most probably the fish-bones were concealed in the blood in the intestinal canal.

Among the other freaks of fish-bones one or two are worthy of mention. Morell Mackenzie[1] records a remarkable case which he saw some years ago with Dr. Turtle at Woodford. A fish-bone had accidentally found its way into an infant's throat, and a very careful examination failed to discover it. The infant wasted and died at the end of a few months. It was then found that the fish-bone had passed through an intervertebral substance and wounded the cord.

In the following case a fish-bone was instrumental in

[1] 'Diseases of the Throat and Nose,' vol. ii, p. 192.

causing intestinal obstruction. In the museum of the Royal College of Surgeons of England is a very interesting specimen (No. 2569), taken from a case under the care of Mr. Coulson. It shows an annular stricture of the rectum six inches above the anus and a small piece of fish-bone sticking in its inner ulcerated surface. The gravid uterus pressed on the foreign body, causing great irritation and effusion of lymph, and complete occlusion of the bowel resulted. The patient was a woman, thirty-four years of age, in good health and more than four months pregnant, who was seized with sickness, constipation, pain, and distension of the abdomen. Fæcal vomiting supervened with more distension and continued constipation. Injections were immediately expelled, and death resulted on the third day from the commencement of the attack.

The preceding remarks will suffice to introduce the subject, and I now append the particulars of my own case.

R. B—, a badly-nourished boy, æt. 9, with glandular enlargements, was admitted into the London Hospital on November 14th, 1882, under the care of Dr. Sutton.

On November 8th, that is to say six days previously, he was eating plaice, and swallowed a small bone. He ran into the yard, followed by his mother, who put her finger down his throat and made him vomit. It was thought that the fish-bone had been ejected, but as pain continued he was taken to a neighbouring doctor, who advised him to go to the hospital. This advice he carried out the next day. In the receiving room of the London Hospital he was seen by the house surgeon and a member of the staff. Saliva was freely dribbling from the mouth. After a careful examination of the mouth and throat a probang was passed, and as the passage was clear he was sent home. Not being relieved he came back to the hospital as a medical out-patient, and was then admitted as an in-patient. His symptoms were pyrexia, stiffness of the neck, œdema of the upper eyelids, profuse salivation,

and a small tender lump on the left side of the neck opposite the cricoid cartilage. When examined the following day he was in the same condition. His pulse was 120, his temperature 101·3,° and his respirations 22. The tenderness and rigidity of the neck continued, but he could not swallow solid food. On the 17th it was noted that the patient was very drowsy, that blood flowed from the mouth, and that the sound of the voice was thicker than usual. He complained of earache. He had two attacks of hæmorrhage on the 17th. On the 18th he was easy. Saliva still flowed from his mouth. The pulse was 128, and the temperature 98°. There was no hæmorrhage. On Sunday, the 19th, hæmorrhage suddenly supervened. Blood flowed in a stream from the patient's mouth, and was received into a spittoon holding a pint. The blood half filled the vessel. Mr. E. H. Fenwick, then house surgeon, now assistant surgeon at the hospital, sent me a note detailing the history and requesting me to see the case. I found the patient in bed lying on his right side, with difficulty in turning round, and the other symptoms previously mentioned. He would not answer questions. Dr. Charlewood Turner saw the patient with me. I came to the conclusion, which I believe Mr. Fenwick had already drawn, that the fish-bone swallowed on the 8th had been arrested in the pharynx, had passed through its walls, and wounded one of the left carotid arteries, that the hæmorrhage proceeded from the wounded vessel, and that it would recur and prove fatal if an operation were not performed. I therefore advised an exploratory operation, and in this advice Dr. Turner concurred. I expected to find the fish-bone, and the wound in the artery, in the situation of the lump in the neck. The patient was taken to the theatre, and chloroform was given by my house surgeon, Mr. Hingston. As I was on the point of commencing the primary incision, my colleague, Mr. James Adams, happened to come into the theatre. He kindly stayed and gave me valuable assistance during the operation.

An incision was made along the edge of the sterno-mastoid for several inches. The muscle was found to be glued to the subjacent parts by recent adhesions. After separating and retracting the muscle, the omohyoid was recognised enclosed in a sheath. Above its anterior belly there was a dark patch about the size of a four-penny piece caused by extravasated blood looming through the fascia. Although it was not absolutely necessary, at Mr. Adams' suggestion, I divided the omohyoid to ensure sufficient room. Having divided the fascia over the large vessels I passed a probe deeply into the cavity which contained the clot, and the left index finger through the mouth into the pharynx, but I could not feel the probe through the wall of the pharynx. I then turned out some clot, and, introducing my finger, ascertained that the probe was in a cavity hollowed out behind the vessels and in the inner side. Having examined the common carotid artery lower down for pulsation I could not very clearly detect any, but once or twice there seemed to be a feeble stream. This indicated that the carotid below the site of the probable wound was blocked with clot, but I deemed it advisable for greater security against hæmorrhage to place a temporary ligature on the artery opposite the divided omohyoid. This was effected with some difficulty owing to the uniform discoloration of artery, vein, nerves, fascia and areolar tissue by the extravasated blood. I could scarcely recognise the structures met with, all being dark and equally stained. I did not see the descendens noni, and though I looked carefully for it I could not distinguish the pneumogastric nerve. Hence it was with some anxiety that I proceeded to turn out more clot from the cavity above for the purpose of finding the wound in the vessel, and applying ligatures above and below the aperture. As this was effected each clot was carefully examined, and in the centre of one the fish-bone was found. Owing to the difficulty in recognising and discriminating one structure from another my colleague suggested that I might include

all in a common ligature, but being anxious to proceed *secundum artem*, and keep the operation free from any avoidable complications, I preferred endeavouring to isolate the artery. More clot was removed and then a free gush of arterial blood took place evidently proceeding from the distal end. Pressure arrested the flow, and the further emission of blood was prevented for the moment by my colleague pulling forward the vessels with a blunt hook. I was then able to find the wounded vessel, and with an aneurism needle to pass a ligature, as I thought, closely round it above and below the seat of injury. Owing to some firm adhesions the upper ligature was passed at a little distance from the wound. In consequence of this necessity—for I had no time to make a prolonged dissection owing to the danger of subjecting the patient to further loss of blood, of the liability to which we were reminded by an occasional jet from the distal end as the hook was shifted or pressure relaxed—I deemed it prudent to divide the artery at the seat of wound to make sure that no branch was given off between the ligatures. When this was done I recognised on the cut section some nerve-fibres, and the question arose whether they belonged to the descendens noni or to the pneumogastric. As they were in front of the vessel, closely adherent, and appeared scarcely numerous enough for the vagus, I came to the conclusion that they belonged to the descendens noni. It will be seen that they belonged to the vagus, which, instead of lying between and behind the artery and vein, took, or had been pressed into, an unusual position in front of the artery, and owing to the inflammation induced by the injury had become firmly adherent to the vessel for some little distance above and below the aperture in the artery. Externally the nerve was stained of the same dark colour as the artery, and only in the centre after section were the white nerve-fibres to be recognised. Believing it to be the descendens noni I made no attempt to disengage it or unite its extremities as I should have done if I had known that it was really the vagus. The

temporary ligature on the trunk of the carotid below was removed, the edges of the wound were dusted with iodoform and approximated, and the patient sent to bed. After the operation he was very restless and thirsty, with difficulty in swallowing. His pupils were equal. He coughed a good deal and vomited two ounces of milky fluid containing coffee grounds. On the 20th his pulse was 140 and respirations 22. He had passed a good night. He took milk, beef tea, and brandy mixture, and was constantly asking for drink. On the 21st he was less restless and more drowsy, with decided weakness in the right arm. Up to the 25th he took his nourishment exceedingly well, but then he began to fail. He lay curled up on his left side with his legs out of bed and his left hand on his left ear and he became very drowsy. He could be roused by opening his eye, and pressing on the conjunctiva, and every now and then he tried to get out of bed. A systolic murmur was heard at the apex. He coughed occasionally but had no return of the vomiting. He was partially paralysed on the right side. He sat up in bed and looked over a picture book on Tuesday, the 28th, but this appearance of improvement was deceptive, for he died exhausted at 1 a.m. on the 29th of November, ten days after the operation. The wound remained healthy throughout. The *post-mortem* was made by Dr. Sutton. The heart and lungs and other internal organs were healthy. On opening the membranes the brain surface in the middle and upper regions of the left hemisphere was seen in two places to be of a green colour and much softened, with pus oozing out. There were two abscesses, each containing green pus, three quarters of an inch and one inch in diameter respectively. The pus was enclosed by a defined boundary, but not by a distinct lining membrane. The surrounding brain was rather softened but not much congested. There was no sign of clotting in the surface vessels of the brain. There was no pus in the left ear. Mr. Hingston removed the pharynx and blood-vessels of the neck, and made a careful examination

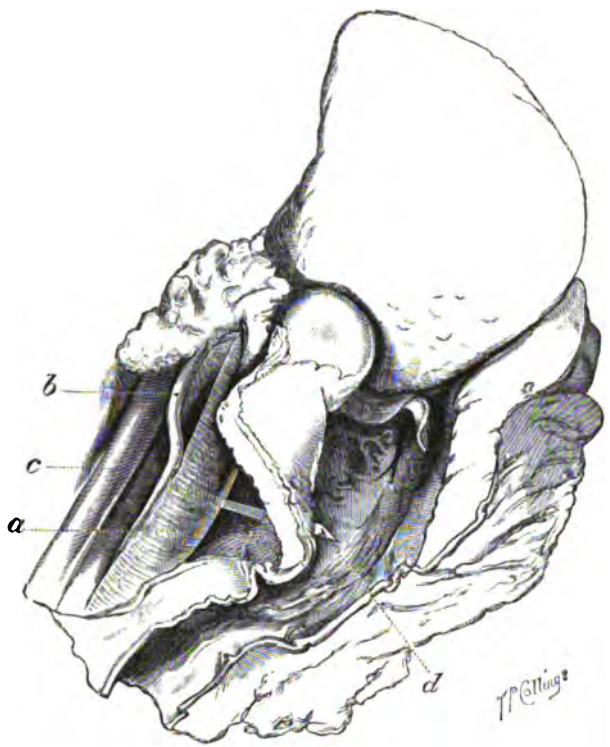
of the parts. The ligature had come away from the upper end of the artery and included nerve, leaving a small round aperture filled with clot. On laying open the vessel the clot was found to be small in quantity, about a quarter of an inch in length, and just sufficient to prevent hæmorrhage. The pneumogastric was adherent for a considerable distance. Some portion of the upper part of it was dissected off the carotid by Mr. Hingston, but more than a quarter of an inch still remains attached thereto. The lower ligature remained round the artery only, and only separated after being cut with the scissors when the artery was laid open. The clot here was abundant, more than an inch long, dark red but decolorized at the tapering end. The small wound in the pharynx made by the fish-bone had contracted and almost closed, but the spot could be recognised by a depression and congestion round it. In the preparation the place of perforation is visible as a small thinned area of mucous membrane with a pin-hole aperture in it situated at the back of the cricoid cartilage and to the left. The seat of the wound in the carotid was three quarters of an inch below the bifurcation. The artery has been laid open. The clot in the lower part of the divided vessels decolorised by the action of the spirit is still present, whilst the scanty clot in the upper part has nearly disappeared. The pneumogastric nerve is seen closely adherent to the upper segment, and looking externally like a branch of the artery. (See woodcut, p. 75.)

Remarks.—Several reflections are suggested by the case itself.

1. The diagnosis was tolerably clear. We had the history of a swallowed fish-bone, the continuance of pain, the visit to the hospital receiving-room with the passage of a probang by which it was rendered probable that the bone had been pushed through the mucous membrane, the local pain and inflammatory symptoms, the pyrexia, and the indications of interference with the carotid artery

and adjacent nerves, viz. the lump in the neck opposite the cricoid cartilage, the œdema of the eyelids, tenderness and rigidity of the neck, inability to swallow solid food, the profuse salivation, the earache on the left side, and, lastly, the attacks of hæmorrhage by which the patient's life was endangered.

2. The diagnosis being established, surgical interference was necessary to prevent death by recurrent hæmorrhage.



a. Left common carotid artery.
b. Pneumogastric nerve, adherent to artery below.
c. Internal jugular vein.
d. Fish bone. This should have been represented as hanging down obliquely and entering the artery at a rather lower point.

3. The operation was undoubtedly a difficult one, partly by reason of the relatively small size of the parts in a patient only nine years of age, but mainly because of the preceding inflammation, formation of adhesions, and that staining of all the tissues of a uniformly dark colour by imbibition of blood which rendered it almost impracticable to distinguish one vessel from another, and, in the absence of pulsation, nerve from blood-vessel. Add to this the necessity imposed upon the operator to ligature the artery as speedily as possible, so soon as the clot which temporarily arrested the hæmorrhage was removed.

Under conditions of this kind it is a great advantage to be able to command the services of a skilled assistant who knows what to do, and does it without instruction, and I acknowledge with pleasure the assistance rendered to me by Mr. Adams.

4. It was suggested by Dr. Sutton from the appearances presented by the abscesses in the brain that they had commenced to form before the operation, and this view is corroborated by the previously existing drowsiness and the difficulty experienced by the patient in turning round in bed. Moreover, clot had abundantly formed at the site of the wound in the vessel and round the fish-bone, and some particles may have been carried up to the brain and arrested in the smaller vessels.

5. I cannot trace any marked ill-effects to the inclusion of the adherent vagus in the ligature, and its subsequent section. Slight cough and some difficulty in swallowing may be attributed to the occurrence, but I do not think that it either determined or hastened the fatal termination. Death resulted from the gangrenous abscesses in the brain, and if these were already in progress prior to the operation, nothing remains but the amount of blood lost at the operation to be placed on the debit side of the account. There were one or two free gushes of blood from the distal side of the wounded artery before it was secured, but whilst admitting the difficulty of an accurate estimate I do not think that more than four or five ounces

were lost during the operation. The loss prior to the operation was far more serious, the patient being as thin and ill-nourished a subject, and as ill adapted for a loss of the kind, as one generally meets with in hospital practice.

6. The slight effects beyond the local paralysis resulting from section of a single vagus, the absence of lung mischief, œdema, and dyspnœa, accord with the results of experiment, and with the negative effects in Mr. Savory's case of "Abscess in the Neck"[1] which, in its course, destroyed a large portion of the carotid artery, jugular vein, and pneumogastric nerve on the left side. Nevertheless, it seems desirable to call special attention to the inclusion of the pneumogastric in the ligature, because the occurrence may furnish a useful hint for future operations. The liability to the formation of adhesions between the vagus and the carotid by inflammatory action set up by a foreign body, and to displacement of the nerve forwards by the pressure of accumulating clot may be usefully remembered by those who are called upon to undertake ligature of the carotid under similar or analogous circumstances.

7. Another point suggested is the danger involved in incautiously passing bougies or probangs for the purpose of clearing the pharynx or œsophagus of a fish-bone or other sharp-pointed body. The history of the case seems to justify an inference that the probang produced the injury to the carotid by pushing the fish-bone through the wall of the pharynx. A similar indictment must be brought against this routine method in Dr. Cresswell Rich's case of perforation of the carotid and in Dr. Waters's case of perforation of the thoracic aorta by a fish-bone. In both, œsophageal bougies or probangs had been employed in the usual manner with aggravation of the symptoms. In Dr. Waters's case a dessert-spoonful of blood was brought up by the patient on the evening of the day on which the probang was passed. The same point might be illustrated

[1] 'Medico-Chirurgical Transactions,' vol. lxiv, 1861, p. 21.

from other cases in which propulsion was attempted. Wagret's case is the most striking. "After a physician had made attempts at the propulsion of the bone, the patient experienced entire relief, and said to his benefactor that he thanked him very much, and that he had saved his life. A few days later the patient died from perforation of the descending aorta."[1]

Improved methods of illumination of the pharynx and œsophagus, the more general use of the laryngoscope and œsophagoscope, exploration with the finger, and the employment of appropriate forceps, may be expected to limit the area within which probangs have wrought mischief. The value of the œsophagoscope invented by Mackenzie is shown by the case which he relates, where at a second sitting he was able to detect and remove from the anterior wall of the œsophagus, about two inches below the cricoid cartilage, a flat lamella of bone about four millimetres square with a small piece of decayed meat adherent to it.[2] At present the chief drawback to the use of the œsophagoscope is the irritation occasioned by its introduction, and this is so pronounced that patients who have once experienced it have declined to submit to it again. For surmounting this obstacle a general anæsthetic is not applicable as it then becomes difficult to place the patient in a favorable position for the illumination of the œsophagus. Better hopes, perhaps, may be entertained of the new local anæsthetic, cocaine, which has already been employed with success in minor operations in the nasal passages, mouth, pharynx, larynx, and rectum, as well as on the conjunctiva. Pending the extraction of the offending body or its passage into the stomach the diet of the patient should be carefully regulated. Hard solid substances should be prohibited and the patient should be restricted to slops, bread and milk, arrowroot, gruel, &c. The exhibition of demulcents like barley water, glycerine, cod-liver and other oils might assist materially in disengaging

[1] Poulet, 'Foreign Bodies in Surgery,' vol. i, p. 93.

[2] Op. cit., vol. ii, p 193.

a small foreign body like a pin or fish-bone clinging to the mucous membrane. If it is necessary to use a probang, the least objectionable and most efficient is the expanding probang or *ramoneur* for withdrawing the body through the mouth. On looking over the cases in the Appendix, and comparing them with each other and with my own case, the following considerations are suggested:

1. The cases which bear the closest resemblance to the one I have related are the cases of Dr. Reid, Mr. Cripps, and Dr. Cresswell Rich, in which the carotid was perforated by fish-bones, and that related by Mr. Bell, of Barrhead, in which a fine sewing needle transfixed the œsophagus and right carotid. In none of these cases, however, was an operation performed.

2. The nature of the foreign body and the size of the vessel injured mainly determine the period at which hæmorrhage appears and death takes place.

(*a*) When the foreign body is very sharp and pointed, or has a sharp pointed projection or a cutting edge, and the artery implicated is the aorta or one of its large branches, death may occur suddenly or in the course of twenty-four or forty-eight hours.

Mr. Colles's patient, a man fifty-six years of age, entered St. Stephen's Hospital on March 30th, 1855. Whilst eating, the patient had experienced a sensation of rupture in the chest, and this pain increased very much during deglutition. Almost immediately afterwards he began to spit blood in large quantity, at first black and then ruddy; the day following the accident he vomited a bone, about an inch long, irregular and with cutting edges, and died the same day at 9 o'clock. A vertical rupture of the posterior wall of the œsophagus was found, corresponding to a rent in the wall of the aorta.

Dr. Hume Spry's patient swallowed a piece of bone. Two days afterwards he was very ill, pale, anxious, and with severe radiating pain, and in the evening he vomited an enormous quantity of blood and fell back on his pillow

dead. The spicule of bone had perforated the œsophagus and wounded the arch of the aorta, and it was found *in situ*.

In other cases an interval of a few days elapses before spitting or vomiting of blood occurs, the fatal issue ranging in its date from six or eight days to two or three weeks from the accident. In one or two cases where the vessel was opened by a gradual process of ulceration the duration of the case has been proportionately lengthened.

(*b*) If the body is rounded and blunt the implication of an important vessel is usually the effect of ulceration, and does not occur, perhaps, for some months after impaction. In the case of Mr. C. L. Bradley's "smasher" the impaction of a counterfeit half-crown in the œsophagus occasioned death from sudden and profuse hæmorrhage from the aorta eight months after the coin had been swallowed, and Mr. Erichsen has recorded an interesting case in which a piece of gutta percha, belonging to a masticatory apparatus was arrested in the œsophagus, and opened a large œsophageal vessel, six months after the patient had swallowed it.

(*c*) Even with a blunt body, however, retained in the œsophagus, fatal hæmorrhage may occur in fourteen or fifteen days. This happened to the unfortunate Corporal M—, who had been in the habit of swallowing six-franc pieces for the amusement of his comrades, usually evacuating them in a few days after a dose of salts. He repeated the experiment once too often, and perished from abundant vomiting of blood on the fifteenth day. The coin was found in the œsophagus, opposite the bifurcation of the trachea, lying on edge between two erosions, one of which communicated with the aorta.

3. Forcible efforts at extraction of the foreign body, or at propulsion into the stomach, may act injuriously in several ways. They may cause the foreign body to scrape or lacerate the mucous membrane, and lay the foundation for subsequent inflammation and ulceration into a neighbouring and perhaps adherent blood-vessel; they may

push the body through the coats of the pharynx or œsophagus and make it penetrate a vessel which otherwise might have escaped injury; they may enlarge a pre-existing laceration of the alimentary canal, and a puncture of a wounded vessel, and lastly they may displace the foreign body from the opening which it is partly plugging and thus hasten the fatal issue.

4. The chief points and symptoms which will assist the surgeon in coming to a right conclusion as to the presence of a foreign body in the pharynx or œsophagus, in determining the situation of the body, and in deciding upon the measures to be taken for its removal, are the following: A definite history of a foreign body having been swallowed; persistence of pain and more or less fixed pain referred to one spot, although radiating twinges may be felt in other directions; dysphagia, and especially continued inability to swallow solids; salivation and dribbling of saliva from the mouth; failure of the foreign body to pass per anum or to be returned through the mouth; expectoration or vomiting of blood, passage of blood by stool, and fainting fits due to hæmorrhage into the alimentary canal. When the foreign body is situated in the neck there will probably be added some local swelling and tenderness or more marked inflammatory signs along the course of the affected vessel.

5. I think it may be concluded that foreign bodies like needles, pins, bristles, and fish-bones which are arrested at the commencement of the œsophagus ought to be capable of extraction by the aid of artificial illumination and forceps, and, failing these, by the *ramoneur*.

Lower down, as at the root of the neck or opposite the arch of the aorta, the continued presence of a foreign body which cannot descend into the stomach under the general means of management indicated above, and which cannot safely be pushed onwards or withdrawn through the mouth, ought to lead the surgeon to the early consideration of the question of an exploratory œsophagotomy. On this subject M. Nevot wrote in 1879 that he believed that

œsophagotomy could render great service in a large number of cases, and he adduced the following instructive instance of its utility :—"On the 14th of February, 1848, M. Lavacherie was called to attend a man named Pascal Dombat, who had swallowed a bone. He practised œsophagotomy with success, found the œsophagus perforated, and the point of the bone in relation with the left common carotid, which was still undamaged. There can be no doubt that in this case the operation rescued the unfortunate Dombat from certain death."[1]

6. The brief duration of many of these cases, their rapid course after hæmorrhage has appeared, and their almost invariably fatal issue, prove the necessity for the utmost promptitude and sagacity on the part of the medical attendant. When hæmorrhage has commenced the life of the patient will hang upon a thread, and the best and only hope of recovery will lie in immediate surgical interference if the wounded vessel can be reached. The services of the surgeon should be sought without further delay, before any considerable quantity of blood has been lost, and before the foundation has been laid for embolism of the cerebral arteries, blood-poisoning, or abscess of the brain, which would nullify all his efforts to rescue the sufferer from impending death.

[1] "De la Perforation de Vaisseaux par les corps étrangers de l'Œsophage," 'Thèse de Paris,' 1879, p. 50.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' New Series, vol. ii, p. 8.)

APPENDIX.

Cases Nos. 1—10, 14, 21, 25, 26, 28, 29, and 42—44 were taken from Nevot's treatise, and the details are quoted from him. The references, however, have been verified and corrected.

I. Perforations of the Aorta.

1. *Wagret*, Obs. de Med. et de Chir., 1718.

Male, æt. 38, swallowed a large bone with a pointed extremity. Rent in mid part of œsophagus and aorta. Bone found in jejunum.

2. *Laurencin* ('Arch. gén. de Méd.,' 1824, t. vi, p. 302).

Male, who had swallowed bone eight or ten days before, entered the hospital with symptoms of left pneumonia. On tenth day vomiting of blood and death in five minutes. Œsophagus perforated, and ulceration of aorta, two inches from the great curvature.

3. *Dubreuil de Brest* ('Journ. Universel,' t. ix, 1818, p. 357).

Soldier swallowed bone. On night of fifteenth or sixteenth day vomiting of bright red blood. Sudden death. Œsophagus and aorta perforated half an inch below the arch. Bone found between œsophagus and aorta.

4. *Revolat* ('Ann. de Soc. de Méd. Prat. de Montpellier,' t. iv, p. 114).

Grenadier swallowed bone; after twenty-one days copious vomiting of blood, and death. Stomach full of blood; two perforations, one at the superior fourth of the œsophagus, and the other towards the cardiac orifice of the stomach. The vessel which had furnished the blood was not looked for.

5. *Lavacherie* ('Mém. de l'Acad. de Méd. Belge,' t. ii, 1848, p. 91).

Male, æt. 18, swallowed a fragment of bone 29th May, 1839. Immediate catheterism discovered nothing. Six days later nausea and vomiting of blood. A small fragment of bone found in a vomited clot. June 10th, two fainting fits. June 16th, abundant vomiting of blood, lasting for two hours and followed by death.

6. *Hugues* ('Lyon Médicale,' t. v., 1870, p. 552).

Male, æt. 32, swallowed a flat triangular bone, apparently without knowing it. Some days after he came into hospital complaining of some ill-defined malady. Fourteen days later vomiting of blood and

hæmorrhagic stools. Death next day. Perforation of œsophagus and aorta. Bone found *in situ*.

7. *Théron* ('Gaz. des Hôpitaux,' 1862, p. 182).

Male, æt. 22, swallowed something, probably a fish-bone. Ulceration of œsophagus followed at length by that of the aorta. Duration of case some months.

8. *Stetter* ('Langenbeck's Archiv,' Bd. xxii, 1878, p. 959).

Male, æt. 26, swallowed bone in soup. Sharp pain, which swallowing increased. Œsophageal sound introduced. At fifth attempt it was pushed strongly and penetrated into the stomach. The patient said he no longer felt anything. Three days later he returned complaining of pain between the shoulders. The sound passed easily, only causing pain in the middle of the œsophagus. Patient went into hospital and left nine days later, saying he was quite cured. He returned to work. Three days later copious hæmatemesis and death in five and a half hours. Double perforation of the œsophagus at level of bifurcation of the bronchi extending from right to left, and very small perforation of the descending aorta 2½ centimetres from the left subclavian. An angular thin piece of bone was found there. Stomach and intestines filled with blood.

9. *Miennée* ('Gaz. des Hôpitaux,' 1851, p. 89).

Soldier, æt. 25, swallowed bone whilst eating soup. Pain in deglutition. Seven days after, vomiting of red frothy blood and bloody stools. Death in the evening. Perforation of the œsophagus and aorta below the arch by a flat triangular piece of bone 3 centimetres in diameter.

10. *Haurmin* ('Rec. de Méd. Mil.,' 1825, t. xvi, p. 245).

Male swallowed piece of bone an inch long, eight or ten days before going into the hospital. There were symptoms of left pneumonia, and a painful spot behind near the vertebral column. On the tenth day he was seized with cough, vomited blood, and died in five minutes. In the middle of the chest there was an opening in the œsophagus as large as a twenty-sous piece, and an ulceration of the aorta two inches below the arch. A small bone an inch in length, with a pointed extremity, was found to the right of the aortic opening.

(N.B. This case is certainly identical with Laurencin's above given.)

11. *C. Laurence Bradley* ('Med. Times and Gazette,' vol. ii, 1868, p. 447).

Male, æt. 21, swallowed a counterfeit coin. This was followed by vague pains in the chest and other symptoms, which were regarded as dyspeptic. He also had a slight cough without expectoration. Eight months after the coin had been swallowed death occurred from sudden and profuse hæmorrhage from the aorta.

12. *Martin* ('Recueil de Médecine Militaire,' t. xx). Poulet, (translation) vol. i, pp. 75 and 94.

Corporal M— had several times swallowed six-franc pieces for the amusement of his comrades, evacuating them in a few days after a dose of salts. He repeated the experiment, and perished from abundant vomiting of blood on the fifteenth day. The coin was found in the œsophagus opposite the bifurcation of the bronchi, lying on edge between two erosions which communicated with the aorta.

13. *Lancet*, Nov. 24th, 1877, p. 789.

In November, 1877, Dr. White, City Coroner for Dublin, held an inquest at the Richmond Lunatic Asylum on the body of an inmate named Nolan, aged forty-seven, who had died suddenly in that institution. After the evidence obtained, the jury found that the deceased came by her death in consequence of hæmorrhage from a punctured wound in the aorta caused by a sewing needle which she had swallowed. Part of the sewing needle was found embedded in the œsophagus, covered with rust.

(In Poulet's work, vol. i, p. 91, Nolan is called Volon and the sex is changed.)

14. *M. Denonvilliers* ('Bull. Soc. de Chir.,' t. vi, 1856, p. 349).

Male swallowed in jest a five-franc piece. The foreign body caused ulceration of the œsophagus and perforation of the aorta. Copious vomiting of blood carried off the patient. The coin was found resting partly in the œsophagus and partly in the aorta.

15. *Duncan* ('Northern Journal of Medicine,' 1844, vol. i, p. 15).

Male, æt. 22, swallowed in his sleep a gold plate carrying some artificial teeth. He suffered from dysphagia, fixed pain, and expectoration of small quantities of blood. Soon after the accident he consulted Mr. Syme, who detected the foreign body in the œsophagus with a probang, and subsequently, when the patient had been removed to the hospital, made an attempt to draw it up with threads passing through the bulb of the probang. Nothing was detected or removed by this manœuvre, and the patient experiencing considerable relief it was believed that the plate had found its way into the stomach, and it was considered inexpedient to make any further

examination. Ten days after the accident the patient vomited the tooth-plate, but a few minutes afterwards expired from hæmorrhage. An ulcerated perforation, communicating with the arch of the aorta, half an inch below the origin of the left subclavian artery, was found in the anterior wall of the œsophagus.

16. *Hume Spry* ('Path. Trans.,' vol. xix, p. 219).

Male swallowed a sharp spicula of bone. Two days afterwards he was very ill, pale, anxious, and with severe radiating pain, and in the evening he vomited an enormous quantity of blood and fell back on his pillow dead. The spicula of bone had perforated the œsophagus and wounded the arch of the aorta and it was found *in situ*.

17. *William Colles* ('Dub. Quart. Jour. of Med. Science,' 1855, vol. xix, p. 325).

Male, æt. 56, entered Steeven's Hospital on March 30th, 1855. Whilst eating, the patient had experienced sensation of rupture in the chest and this pain increased very much during deglutition. Almost immediately afterwards he began to spit blood in large quantities, at first black and then ruddy. The day following the accident he vomited a bone about an inch long, irregular, and with cutting edges. He died the same day at 9 o'clock. Blood was found in the pleura, pericardium, and posterior mediastinum, blood in the stomach and small intestines, and a vertical rupture of the posterior wall of the œsophagus half an inch long corresponding to a rent in the wall of the aorta.

18. *Ramskill* ('Lancet,' 1871, i, p. 646).

Male swallowed a fish-bone which lodged in his throat. He went at once to the London Hospital, but returned without having had it removed. On reaching home he took to his bed, and complained of pain in his chest. He soon afterwards felt sick and began to retch without actually vomiting. The day before admission, feeling somewhat better, he sat up for a couple of hours, but on returning to bed felt much worse and complained of great pain across the region of the stomach. He passed a very restless night, and in the morning whilst coughing vomited a quantity of dark-coloured coagulated blood, amounting to three quarts, according to the estimate of his friends. He was taken to the hospital and admitted under Dr. Ramskill, but died the same evening, after bringing up a great quantity of arterial blood together with blood-clot. At the post-mortem examination Dr. Sutton found at the level of the fourth dorsal vertebra two perforating ulcers in the œsophagus; one on the left side communicated with the aorta by an opening which admitted a

probe, whilst the other had extended through the œsophagus and caused thickening round the vena azygos, which was plugged with blood-clot.

19. *Museum of St. Bartholomew's Hospital* (Catalogue).

No. 1376 is a preparation showing a ragged laceration of the aorta beyond the origin of the left subclavian involving more than half its circumference. It was taken from the body of a middle-aged man, who after eating some fish complained of constant pain behind the first bone of the sternum. Every day he spat up blood, for the most part bright red, sometimes dark, and a large quantity passed per anum. He died from exhaustion. At the post-mortem a lance-shaped fish-bone was found transfixing the œsophagus and the arch of the aorta. It was evident that the lacerated wound of the vessel had been produced by the movement of the artery as it pulsated on the point of the fish-bone.

20. *Auvert* ('Selecta Praxis Med. Chi.,' Paris, 1851).

Male swallowed a fish-bone. All the symptoms of a foreign body, and some expectorations of blood. On the third day copious hæmorrhage carried off the patient. Anterior wall of œsophagus perforated and the aorta near the arch.

21. *Bousquet* ('Bull. de la Soc. Anat.,' 1877, p. 317).

Soldier entered the hospital for pleuro-bronchitis of six days' standing, on 20th March, 1877. On the 11th April, he asked to be allowed to go out, but his medical attendant declined. Next day he had vomiting of blood, and bloody stools. He died on the 13th. The œsophagus and aorta were both perforated, and the former contained a sharp-edged fish-bone 2 centimetres long.

22. *Dr. Waters*, Liverpool Royal Infirmary, 1879. Communicated by Mr. Paul.

Mary Hazelton, æt. 55, swallowed a fish-bone, which became impacted in the œsophagus, four days before her admission into the Royal Infirmary. On admission, 26th November, 1879, she complained of great pain in the chest, opposite the lower end of the sternum. Deglutition was very painful and difficult. An œsophageal bougie was passed without a hitch, but she brought up a dessert-spoonful of blood the same evening. Nov. 29th, temp. 103·4°, dulness and tubular breathing in the interscapular region. Nov. 30, 8 p.m., temp. 104·8°. At 11 p.m., a sudden, small hæmorrhage from the mouth followed by death, almost immediately, from syncope. *Post-mortem examination.*—Stomach and duodenum distended with blood-

clot, weighing 2¼ lbs., and forming an accurate cast of their cavities. Just at the junction of the transverse with the descending part of the arch of the aorta was a perforation that would have admitted a No. 10 catheter; the opening passed into a fœtid, inflammatory swelling between it and the œsophagus and surrounding the parts about the roots of the lungs, accounting no doubt for the dulness noticed in the interscapular region. The opening passed directly through this fœtid cellulitis into the œsophagus, where it was large enough to admit the little finger. No fish-bone could be found. Probably it had been washed away in the gush of blood.

23. *Aschenborn* ('Berliner klin. Wochens.,' 1877, t. xiv, p. 725), 'Lond. Med. Record,' vol. vi, 1878, p. 21, quoted by Durham, in Holmes and Hulke's 'System of Surgery,' vol. i, p. 787.

A young man swallowed a hard morsel of bread containing, apparently, a needle two inches long. The œsophagus was penetrated and the aorta transfixed. Blood was passed by stool on the ninth and tenth days, and the patient succumbed in a few minutes on the eleventh day from a copious hæmorrhage from the mouth.

II. Perforation of an Undetermined Artery.

24. *Erichsen* ('Science and Art of Surgery,' 8th ed., vol. ii, p. 661).

Male swallowed a piece of gutta percha, part of an artificial masticatory apparatus. A few days after examined by a surgeon, who could not detect any foreign body. Inability to swallow solids. Six months later examined by Mr. Erichsen, who failed also to discover the body. One day while at dinner the patient suddenly vomited a large quantity of blood, and fell down dead. The gutta percha had formed for itself a bed in the wall of the œsophagus, and lay parallel with the inside of the tube. The œsophageal vessel opened was not ascertained. The carotid arteries and jugular veins were not implicated. The surface of the gutta percha, which looked towards the œsophagus, being constantly covered and smoothed over by mucus, and protected by a rim of swollen mucous membrane, had allowed the probang to glide smoothly over it.

III. Perforation of Œsophageal Artery.

25. *Monestier* ('Bull. de la Soc. Anat.,' vol. viii, 1833, p. 226).

Young female eating cabbage, swallowed a piece of the vertebra of a pig. This caused a slough involving an œsophageal artery.

On the separation of the slough slow effusion of blood took place into the stomach, which relieved itself from time to time by vomiting and stool. The patient died suddenly at the end of three weeks.

IV. Perforation of Inferior Thyroid.

26. *Pilate* ('Bull. de la Soc. Anat. de Paris,' 1867, p. 648).

Female, æt. 55, swallowed a piece of bone; slight pain in swallowing. Eight days later she entered the hospital. Soon after hæmatemesis and frequent and copious bloody stools. Death in a short time. A piece of bone 3 centimetres long and 3 millimetres broad, with one end pointed, lay horizontally across the œsophagus at the inferior border of the cricoid cartilage. The lateral walls of the œsophagus were perforated and the adherent thyroid gland formed the base of the œsophageal ulcerations. One of the branches of the right inferior thyroid was involved.

V. Perforation of Carotid.

(*a*) *Left Carotid.*

27. *Bégin*, quoted by Dr. James Duncan, 'Northern Journal of Medicine,' vol. i, p. 20.

Male, while eating soup, swallowed a piece of bone, which stuck in the œsophagus; attempts to push it on towards the stomach were made and appeared to be successful. No further inconvenience was experienced till a month later, when he had sharp pains on the left side of his neck which continued with slight intermissions for some time. Everything seemed to be going on well, when he suddenly threw up large quantities of blood, perhaps to the amount of several pounds. The hæmorrhage presently ceased, but the next day it returned and proved fatal. On examining the body there was found in the œsophagus, about its upper third part, two parallel ulcerations, that on the right side nine lines in breadth, that on the left twelve; opposite the latter there was an adhesion between the œsophagus and the corresponding part of the carotid. In this vessel erosion had produced a small opening, about a line in diameter, which proved to be the source of the hæmorrhage. In all probability the ulcerations were due to scraping or tearing the mucous membrane during the operation of pushing the bone into the stomach with a probang.

28. *Auvert*, op. cit.

Perforation of œsophagus and left common carotid. Death.

29. *Dumoustier* ('Recueil de Méd. Militaire,' 1828, t. viii, p. 231). Male swallowed a beef-bone while eating soup. He entered the hospital on 18th April, 1820, complaining of sharp pain in the upper third of the œsophagus. Attempts at propulsion were made, great improvement followed, and patient left on the 18th of May. He came again on June 14th and stayed a few days. On 18th July he again returned; since accident he had experienced pain at anterior part and left of neck. No fresh symptom till 27th, when copious hæmatemesis occurred, recurring on 28th; he died on the 29th. At the upper third of the œsophagus were two parallel ulcerations, and there was a small hole in the left carotid united to the œsophagus by adhesions.

30. *Reid* ('Ed. Med. and Surg. Journal,' vol. xlviii, 1837, p. 95). George B—, æt. 27, tailor, was eating fish when a bone was arrested in his throat. The following day, he saw a surgeon who did not think there was any bone in the case, but attributed the pain and irritation to inflammation of the parts brought on by a fit of intemperance. At this time there was much pain and some tumefaction in the throat, and the patient could not swallow his spittle, which flowed from the angle of his mouth into a cup as he lay on his side. The next day he was twice bled to a soup-plate full, and on the fourth day was blistered over the sternum. On the fifth day there was tumefaction over the whole of the cervical region and he was bled again to a soup-plate full. On the eleventh day he was sick and vomited about a pint of fluid blood, not in the least coagulated. The sickness and vomiting of blood recurred the following morning. At 5 a.m. on the thirteenth day he awoke from sleep very sick, and just as he was about to get a cupful of tea he gave a groan and immediately expired, without external symptoms of hæmorrhage. At the post-mortem the stomach was found filled with blood. An inch above the left sterno-clavicular articulation two slightly ulcerated openings were found on each side of the tube. The left carotid adhered to the œsophagus and had in it a longitudinal opening to the extent of a quarter of an inch. The right carotid was sound. The fish-bone was not found.

31. *H. C. Johnson*, Durham, op. cit., p. 745.

Boy, æt. 7, sustained a penetrating wound on the left side of the pharynx, through falling whilst he held the sharp end of a parasol in his mouth. The point was thrust so forcibly backwards that it nearly made its appearance through the skin at the side of the neck. Considerable hæmorrhage took place at once, and recurred at night. About the 7th or 8th a slough came from the interior of the mouth,

and arterial hæmorrhage to the extent of five ounces, and was arrested by external pressure. Soon afterwards the boy was admitted into St. George's Hospital, and a fluctuating swelling as large as half a hen's egg below and behind the left ear was opened, giving exit to pus and blood-clot. Two days later a gush of arterial blood followed a fit of coughing. Mr. H. C. Johnson tied the common carotid. No further hæmorrhage occurred, and the patient was discharged cured twenty-seven days after the operation.

32. *Dr. Cresswell Rich* and *Mr. Paul*, Liverpool. Preparation in museum of Liverpool School of Medicine.

Boy, æt. 6, had fluke for dinner on February 23rd, 1883. An hour afterwards he complained of something sticking in his throat. He was taken to a dispensary and told that the bone had been pushed down by an instrument. He continued unable to eat solids. Five days after the accident castor-oil was given to him, and an hour after taking it he vomited clotted blood. He was taken to the Infirmary, vomiting blood all the way. On reaching the hospital he was in a faint, the surface of the body and the face being livid and blue. Ergotine was subcutaneously injected. He became alternately conscious and unconscious and continued to vomit blood at intervals till death took place on the following day.

Post-mortem examination.—Well-nourished boy. On anterior wall of gullet, opposite the commencement of the trachea, there was a perforation of the size to admit a No. 8 catheter. It was circular, had a punched-out appearance, with perpendicular edge raised inside, and of a purplish red colour. There was neither discoloration of the surrounding mucous membrane nor undermining or separation of the coats of the œsophagus. There was no adhesion between the gullet and the left common carotid artery, but there was an opening in the vessel of the same size as that in the gullet. The vein was not injured. All the organs were very anæmic. No fish-bone or other foreign body was found; it had probably been washed away in a gush of blood. The mucous membrane of the alimentary canal was healthy, and there was no sign of any hæmorrhage from it. The large bowel was full of altered blood.

33. *Rivington*, 'Med.-Chir. Trans.' (Case described in present paper.)

(*b*) *Not stated, but probably Left Carotid.*

34. *Cripps* ('Lancet,' 1878, vol. i, p. 834).

In the discussion at the Clinical Society on the 24th May, 1878,

on Dr. McKeown's paper on a successful case of œsophagotomy for the removal of a set of artificial teeth from the œsophagus, impacted at the lower part of the neck, Mr. Cripps related a case in which a small fish-bone had been swallowed. Some pain was felt for a week, but no other inconvenience, when suddenly a short time after severe pain occurred, followed by a gush of blood from the mouth and rapid death, which was found to have been due to the bone having perforated the œsophagus and caused ulceration of the carotid at its bifurcation.

35. *Fingerhuth*, quoted by *Mackenzie*, 'Diseases of Throat and Nose,' vol. i, p. 109. Quoted also by Durham, op. cit., p. 784.

A piece of tobacco pipe was lodged in the side of the pharynx, and after an interval of eight months occasioned fatal hæmorrhage by wounding the carotid in a sudden movement of the head.

(*c*) *Left Ascending Pharyngeal.*

36. *Mr. Morrant Baker* ('St. Bartholomew's Hosp. Reports,' vol. xii, 1876, p. 163).

Man, æt. 23, fell with a clay pipe in his mouth. Two days afterwards he applied at St. Bartholomew's Hospital for sore-throat. The case was at first thought to be medical, but was subsequently transferred to the house surgeon as a case of abscess of the tonsil. The supposed abscess was punctured and only blood escaped. In the evening several more ounces of blood escaped from his mouth. Two days afterwards nearly a pint of blood was lost and a cavity found in the left side of the pharynx was plugged. The next day hæmorrhage recurred, and on examination under anæsthesia a piece of tobacco pipe three quarters of an inch long was found in the tonsil. This was removed and the cavity plugged. The common carotid was then tied, but the patient died in three hours. At the post-mortem an irregular cavity was found above and behind the left tonsil. The internal carotid lay about one eighth of an inch away from the cavity and had not been wounded. Into the cavity no artery could be traced, but the ascending pharyngeal appeared to terminate abruptly just at its edge and was stained by perchloride of iron.

(*d*) *Right Carotid.*

37. *Bell, of Barrhead* ('Lond. Med. Gaz.,' n. s., vol. i, 1843, p. 694).

Lad, æt. 18, swallowed a sharp body (as he thought, a pin) whilst he was eating some oatmeal porridge, and felt it sticking in his throat. He began to spit blood on the ninth day at 6 p.m., and at

11 p.m. brought up a soup-plate full. He kept spitting up mouthfuls till the next morning, when he vomited a large quantity, and died. The œsophagus was transfixed opposite the middle of the thyroid cartilage by a fine sewing needle three inches long, its point resting against the right common carotid artery. The walls of the vessel were destroyed, and a considerable opening, communicating with the œsophagus, had been made in the vessel, the internal coat of which had disappeared for one and a half inches, and was quite rotten. An ounce of pus and blood was found between the œsophagus and the artery.

(e) *Both Carotids.*

38. *Guthrie* ('Wounds and Injuries of Arteries,' p. 77).

A soldier swallowed an instrument composed of two half phial corks, fastened together with strong thread and with three pins thrust through each, so that the pins projected on each side. This machine became entangled at the commencement of the œsophagus, and caused death from hæmorrhage after the lapse of some days. The patient at first complained of some difficulty of breathing and uneasiness in the chest. The fauces became slightly reddened and inflamed and he was utterly incapable of swallowing anything but liquids. This was followed by ptyalism and soon by spitting of blood of a light scarlet colour, without any cough; increasing in quantity daily, until he brought up six or eight ounces. A day or two afterwards the blood poured out of his mouth so rapidly that Guthrie was sent for. He arrived in time to see the blood fill a chamber-pot, when the patient fell back, dead. The instrument rested across the œsophagus so that the points of the pins were close to the carotid arteries, and having by degrees given rise to ulceration of the œsophagus, wounded them on both sides. Every elongation or pulsation of the arteries had brought them against the point of one or more of the pins, the marks of which were observable in several small holes of different sizes on the sides of the vessels. As one or two of these became larger from the constant attrition, blood came through into the œsophagus, and as they again increased by ulceration, larger holes were formed from which the sudden and fatal hæmorrhage took place. Guthrie adds, "The instrument and the arteries I sent from North America to the late Dr. Hooper, and they ought to be in the museum of King's College."

VI. Perforation of Right Subclavian (abnormal).

39. *Kirby* ('Dublin Hospital Reports,' t. ii, p. 224).

A poor woman, one of those miserable creatures who feed in the streets of Dublin upon the mixed offal which they receive from servants, was greedily enjoying such wretched fare, when a morsel stuck in the œsophagus. She was taken to St. Peter's and St. Bridget's Hospitals, but died before Mr. Kirby arrived. Tracheotomy and artificial respiration were of no service. At the post-mortem two large morsels of food were found in the œsophagus, one below the cricoid cartilage and the other as low down as the upper extremity of the sternum. The latter morsel contained a piece of bone, an inch and a half long, one of its ends being sharp and pointed. The bone lay obliquely across the œsophagus, transfixing it at its left and posterior part, and wounding the right subclavian artery, which, contrary to its usual course and origin, lay in this situation as it passed from the left of the arch of the aorta, where it arose towards the right shoulder. The surrounding cellular tissue was filled with blood, which, accumulating principally at the sides of the neck, had produced a remarkable fulness there noticed during the previous examination of the patient.

VII. Perforation of Pulmonary Artery.

40. *Bernast* ('Jour. hebd. des Sci. Méd.,' 1833, also 'Lond. Med. Gazette,' May 11th, 1833, p. 175, and Duncan, op. cit.).

A young soldier swallowed a sharp bone while taking soup. He entered the Toulon Hospital, continued in great pain for some days, and threw up some ounces of blood. He died on the eighth day. A flattened sharp-pointed bone was found in front of the œsophagus, which it had perforated, and there was a minute opening in the pulmonary artery at its bifurcation. A large quantity of extravasated blood was found in the chest.

VIII. Perforation of Heart and Right Coronary Vein.

41. *Andrew* ('Lancet,' vol. ii, 1860, p. 186).

A woman was found on a doorstep in a dying state, and taken to University College Hospital. The previous history could not be gathered. At the post-mortem it was found that a fish-bone had penetrated the stomach close to the œsophagus, then the diaphragm and pericardium, and the posterior surface of the heart, and finally inflicted a jagged wound in the middle of the septum immediately over the right coronary artery and vein, penetrating the latter vessel. The pericardium was filled with a pint and a half of fluid blood.

IX. Perforation of Demi-Azygos Vein.

42. *Saucerotte* ('Ann. de la Soc. de Méd. pratique de Montpellier,' t. ii, p. 247).

Carbineer swallowed a piece of bone. Sharp pain towards cardiac orifice. Eight days afterwards Saucerotte introduced a wax bougie. The bone was dislodged and returned by vomiting with much blood. Death next day. The œsophagus was divided vertically for 3 centimetres at the level of the sixth rib, and a large vein, believed by Saucerotte to be the demi-azygos, was implicated.

X. Perforations of Vena Cava, Superior and Inferior.

43. *Laurent Lovadina* ('Jour. Complém. du Dict. des Sciences Médicales,' t. i, 1818, p. 93).

Male, æt. 42, swallowed a bone, which was arrested at the back of the throat and required much time and effort to make it descend into the œsophagus. Angina, sharp pains at each respiration, and efforts at vomiting persisted for ten days, when the patient, whilst raising himself to make water, was seized suddenly with vomiting of blood and expired.

Autopsy.—Great gangrenous patches upon the soft palate, pharynx, and œsophagus. A little below the orifice of the gullet there was a great rent, which was thought to have been produced by the sharp angles of the bone. On the outer and towards the anterior part of the vena cava superior was a rent an inch long and about an inch from the right auricle. Another less extensive rupture was found on the anterior face of the vena cava inferior before its entry into the pericardium.

44. *Coester* ('Berliner klin. Woch.,' 1870).

Male, æt. 56, complained on Nov. 11th of great pain radiating from the epigastrium, loss of appetite, and oppression. Castor-oil gave some relief. On the 17th the painful crisis returned, followed by vomiting of blood and sudden death. The pleura and stomach were found filled with blood. The œsophagus was perforated half an inch above the diaphragm. In the perforation a rather large pointed and cylindrical piece of bone was engaged. The descending cava had contracted adhesions to the œsophagus and was perforated like it.

45. Dr. H. Thompson ('Brit. Med. Journal,' 1874, vol. ii, p. 571), quoted in text, p. 65.

SCARLATINAL ALBUMINURIA; AND THE PRE-ALBUMINURIC STAGE,

STUDIED BY FREQUENT TESTING.

BY

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I PURPOSE giving in the following paper a detailed account of observations conducted upon 180 consecutive cases of scarlet fever in the wards of the City of Glasgow Fever Hospital. The ages of the patients ranged from two to thirty-five years, the great majority (84 per cent.) being under fifteen years of age. Of the cases examined twelve died from all causes. The period of observation extended over one year (1882-83) and involved the examination of upwards of 35,000 specimens of urine. Three specimens of urine from each case under observation were examined daily from the day of admission till dismissal from hospital. The minimum period of residence imposed by the sanitary authorities was fifty-six days,[1] calcu-

[1] Patients were occasionally dismissed a day or two before the completion of their term, but more frequently they were kept beyond it.

lated from the first appearance of fever. In a few chronic cases the investigations extended over a period of from five to six months. Careful notes of the condition of the urine were made daily till all traces of albumen and blood-colouring matter had disappeared; in one or two instances, however, the patient was dismissed before the return of the urine to its normal condition. The samples were collected at 6 a.m., before breakfast; at 12 noon, just before dinner; and at 8 p.m. In this way the slightest trace of any abnormal constituent could be detected within a few hours of its appearance. Every precaution as regards the cleanliness of vessels was taken to ensure accuracy in the results. To eliminate as fully as practicable errors arising in individual cases from so-called "alimentary" albuminuria, the diet was made uniform for each stage of the disease. The same object was kept in view when the above-mentioned hours were selected for collecting the urine. When thought necessary specimens of urine were examined every three or four hours; such cases were, however, exceptional.

The special interest of the investigation centred round the detection of minute quantities of blood-colouring matter, of albumen, and of organic deposits of renal derivation. For the detection of the first of these I for some time employed both the spectroscope and the guaiacum test, but soon gave up the former on account of the difficulty attending the detection by its means of very minute quantities of blood in turbid urine. The difficulty is not diminished when we turn to the microspectroscope, for although with it a single red corpuscle will give the characteristic bands, yet the time necessarily expended in the search is too great for ordinary purposes. The guaiacum test on the other hand is exceedingly delicate, simple, convenient, and reliable.[1]

[1] The method employed in using the guaiacum test was that usually followed in the Glasgow School of Medicine (see 'Finlayson's Manual'):—To a few drops of urine from the bottom of the urine-glass a drop of tincture of guaiacum is added; ozonic ether is then gradually poured into the tube until

In testing for albumen, nitric acid in the cold was chiefly relied upon on account of its convenience and the rapidity with which a large number of specimens could be tested in a comparatively short time. This test was applied by a pipette very much in the same way as in the case of the picric acid test described below. Before this inquiry was begun I had, while resident assistant in the Glasgow Western Infirmary, had ample opportunities for studying the nitric acid test for albumen and all its well-known fallacies. In cases where there was any doubt the testing was checked by boiling with the after-addition of acetic acid and also by the use of the picric acid test. Of these tests picric acid is the most delicate, and nitric acid in the cold seems to be inferior, as a rule, to the boiling test. The best results were obtained with picric acid when the urine to be tested was allowed to flow from a pipette, the point of which rested on the bottom of a test-tube containing a quantity of a saturated solution of the acid, so that it fell to the bottom without much admixture. The result was confirmed by boiling.

While working at this subject I instituted a series of comparative experiments of specimens of presumably normal urine, and in but few instances did I detect an appearance which could be readily confounded with that caused by albumen; yet it must be confessed that picric acid shares with the other two tests the peculiarity of causing, under certain circumstances, a precipitate (mucin ?) very like that due to albumen. In most cases this cloud is at a little distance from the contact-surface, but occasionally the resemblance is so misleading that it might be best to reserve the picric acid test for a confirmation of the other tests or for demonstrating negative results. In certain cases when nitric acid in the cold and the test by boiling failed to detect albumen, picric acid gave the

the precipitate formed by the action of the urine on the guaiacum is completely dissolved. If blood be present a blue colour varying in intensity is developed. This seems to me the most delicate method of using the guaiacum test.

characteristic reaction, and its correctness was in most cases confirmed by evaporating the urine to a small bulk and employing the first two tests when each gave confirmatory results. Throughout the investigations I assumed, in any doubtful case, that albumen was present in a specimen of urine when characteristic appearances were got with all these tests.

I will discuss the subject under the following heads :

I. The period of occurrence of albuminuria in scarlatina.

II. The frequency of albuminuria in scarlatina.

III. The relations which blood and albumen bear to each other in the urine of scarlatinal nephritis.

IV. The so-called "pre-albuminuric stage" in scarlatinal nephritis.

V. Treatment.

I. Period of Occurrence.

For purposes of convenience it will be best in discussing this subject to divide the cases into two classes :

1. Cases of what may be called "Initial Albuminuria."

2. Cases of "Late Albuminuria."

Whether these two classes are due to the same pathological changes in the kidney, or whether the first is due to the primary febrile disturbance, and the second to recognisable, though it may be minute, vascular and cellular changes in the kidney, is a subject which, should opportunity offer, I hope to investigate further. In the meantime the various periods at which this complication of scarlet fever most frequently occurs will occupy our attention.

1. In the first class are included all those cases in which albumen was detected in the course of the first week of the illness; in the second those in which it appeared at a later period, after the primary scarlatinal symptoms had begun to subside. This subdivision is justifiable on the

ground that patients with scarlet fever frequently suffer from two attacks of albuminuria, separated by a well-marked interval. No hard and fast line can be drawn between these two classes of cases, and it must be confessed that the distinction as regards their exact period of occurrence is arbitrary. My object in drawing the distinction is to emphasize the frequent occurrence of an interval between the two.

Table showing Duration of the Interval between "Initial" and "Late" Albuminuria.

Number of case in table.	Interval between "Initial" and "Late" albuminuria.	Number of case in table.	Interval between "Initial" and "Late" albuminuria.
No. 10 ...	Days 3 (5th—9th)	... No. 20 ...	Days 5 (7th—11th)
„ 11 ...	„ 10 (8th—18th)	... „ 21 ...	„ 3 (6th—9th)
„ 12 ...	„ 8 (7th—15th)	... „ 22 ...	„ 25 (6th—31st)
„ 13 ...	„ 4 (6th—10th)	... „ 23 ...	„ 3 (8th—11th)
„ 14 ...	„ 9 (7th—16th)	... „ 24 ...	„ 8 (7th—15th)
„ 15 ...	„ 3 (9th—12th)	... „ 25 ...	„ 20 (3rd—23rd)
„ 16 ...	„ 17 (5th—22nd)	... „ 26 ...	„ 12 (5th—19th)
„ 17 ...	„ 4 (8th—12th)	... „ 27 ...	„ 12 (9th—21st)
„ 18 ...	„ 8 (7th—15th)	... „ 28 ...	„ 15 (4th—19th)
„ 19 ...	„ 6 (4th—10th)	... „ 29 ...	„ 5 (9th—14th)

Of cases of "Initial" albuminuria I have no fewer than 40 to record out of a total of 112 cases of albuminuria of all kinds in 180 cases of scarlatina. These cases again admit of subdivision into three classes:

A. Cases running on to "Late" albuminuria without a break—9 cases. (See table, p. 116, Nos. 1—9 inclusive.)

B. Cases followed by "Late" albuminuria after a variable interval—21 cases. (See table, p. 116, Nos. 10—30 inclusive.)

C. Cases not followed by "Late" albuminuria—10 cases. (See table, p. 118, Nos. 31—40 inclusive.)

"Initial" albuminuria does not of itself seem to be a cause for great anxiety, even when the urine is for the first few days loaded with albumen and blood. It is only when it shows a tendency to join hands with "Late" albuminuria that it becomes serious, and it is only then

that one would be inclined to take into consideration the possibility of its bringing about of itself a fatal result. Cases of malignant scarlet fever are no doubt almost invariably complicated with nephritis, and the blood and albumen may be even very abundant, yet the nephritis appears to take a very subordinate part, in comparison with many of the other lesions, in bringing about a fatal termination. I have seen only one case of malignant scarlatina without accompanying albuminuria. This case was peculiar in other respects, and will be noticed later on. (See "Dropsy without Albuminuria;" p. 106.)

Nine out of the 40 cases of "Initial" albuminuria ran on without intermission to "Late" albuminuria. These were all more or less severe, like those of the next class, and in one of the latter the last traces of albumen had not disappeared on the 140th day.

In 21 cases "Late" albuminuria followed after an interval of some days, during which the urine was quite free from albumen or blood.

In 10 cases the "Initial" albuminuria passed off completely, the patient showing no further sign of nephritis after the ninth day of the fever.

2. "Late" albuminuria may come on at any time between the ninth and forty-eighth day, but is much more common at certain periods in the course of the fever than at others, and seems to have a preference for the beginning of the second, third, and, in a less degree, the sixth week.

Table showing the Number of Cases of "Late" Albuminuria, not preceded by "Initial" Albuminuria, occurring at Various Dates of the Fever.

Day of illness.	Number of cases occurring at given date of fever.	Day of illness.	Number of cases occurring at given date of fever.
9th	5	15th	9
10th	4	16th	5
11th	1	17th	6
12th	4	18th	1
13th	3	19th	2
14th	5	20th	1

Day of illness.	Number of cases occurring at given date of fever.	Day of illness.	Number of cases occurring at given date of fever.
21st ...	2	32nd ...	3
22nd ...	2	35th ...	3
23rd ...	1	36th ...	2
24th ...	1	37th ...	1
25th ...	2	39th ...	1
26th ...	1	40th ...	1
27th ...	1	46th ...	1
29th ...	1	47th ...	1
30th ...	1	48th ...	1
31st ...	1		

Table showing the Number of Cases of "Late" Albuminuria, preceded by "Initial" Albuminuria (with an interval between) occurring at Various Dates of the Fever.

Day of illness.	Number of cases occurring at given date of fever.	Day of illness.	Number of cases occurring at given date of fever.
9th ...	2	18th ...	1
10th ...	2	19th ...	2
11th ...	2	21st ...	1
12th ...	2	22nd ...	1
14th ...	1	23rd ...	1
15th ...	3	31st ...	1
16th ...	1		

It will be observed that the numbers cluster about the ninth and fifteenth days. Cases arising at these periods seem the most characteristic, the albuminuria running a course usually of some length and often of great severity. Albuminuria occurring at other periods would appear to last, at most only a few days, and now and again its presence is shown merely as an occasional trace of albumen in the urine.

Illustrations of Very Slight and Transient Albumen or Blood in Urine.

Number on table.	Day of fever.	Total duration of albumen or blood.
46 ...	21st ...	Trace on one occasion.
102 ...	22nd and 23rd ...	On two days only.
82 ...	27th ...	Trace on one occasion.
54 ...	29th till 53rd ...	Trace occasionally.
76 ...	31st till 33rd ...	Trace for three days.
87 ...	40th and 46th ...	Trace on two occasions.

II. Frequency.

Of the 180 cases examined 112 or 63·2 per cent., showed signs of renal affection by the presence of albumen or hæmoglobin *i. e.* blood, in the urine, with or without dropsy, as the case might be. In some cases, however, the evidence of kidney mischief was so slight and evanescent that but for careful and frequent testing the presence of these substances would no doubt have been overlooked.

Two cases, or 1·1 per cent. in the 180, presented *anasarca, without albumen* showing itself in the urine. Sixty-six cases, or only 36·7 per cent. of the whole, escaped entirely.

Of the 112 cases of nephritis 55, or 49·1 per cent., were cases of pure albuminuria, while 57, or 50·9 per cent., came under the class hæmaturia.

Anasarca was observed in only 24 of the 180 cases examined. Of these, 22 suffered from very decided albuminuria, while the urine of the remaining 2 cases did not at any time show the slightest trace of albumen or blood, though these were sought for with the greatest care.

It is perhaps unnecessary to point out that 180 cases form far too narrow a foundation on which to base conclusions as to the probable frequency of the renal affection in any given epidemic of scarlet fever. The above statistics can therefore apply only to that group of cases upon which the investigations were conducted.

III. Relations which Blood and Albumen bear to each other in the Urine of Scarlatinal Nephritis; and Dropsy without Albuminuria.

The abnormal constituents present in the urine of scarlatina patients are not the same in every case. The presence of albumen is of course the principal evidence of

renal disease; but in many cases hæmoglobin is added in varying proportions; and in a few of these last, albumen is *apparently* absent altogether. From this point of view I would subdivide all cases of scarlatinal nephritis as follows:

1. Those cases in which there is albumen from beginning to end without there being at any time the slightest trace of blood-colouring matter in the urine: 55 cases, or 49·1 per cent. (See in table on p. 116 all cases except those referred to in the following two classes.)

2. Those in which blood only seems to be present, and in which the albumen and blood-colouring matter increase and diminish in quantity *pari passu*, so that these constituents seem to be in the same relative proportion as in blood itself. It is in this class of cases that we sometimes find what has been called a "pre-albuminuric stage," and in which there sometimes also exists what might with equal propriety be called a "post-albuminuric stage:" 28 cases, or 25 per cent. (Nos. 16, 20, 22, 27, 40, 41, 42, 43, 44, 45, 55, 56, 58, 64, 65, 70 ?, 76, 77, 79, 83, 86, 90, 92, 94, 96, 99, 101, 103).

3. Those in which we have blood, as in the last class, but in which there is an excess of albumen in addition to that due to the blood. In this class of cases there is no "pre-albuminuric" and usually no "post-albuminuric stage." In a few of the cases which I have included in this class, the excess of albumen seems to disappear, leaving some blood lingering behind, and so giving rise to a "post-albuminuric stage," but in the majority of the cases the albumen appears before, or simultaneously with, the blood-colouring matter, and continues in appreciable quantity after all trace of hæmoglobin has disappeared from the urine: 29 cases, or 25·9 per cent. (Nos. 1, 4, 7, 10, 12, 15, 17, 21, 26, 28, 29, 49, 50, 60 ?, 61, 62, 63, 69, 71, 73, 78, 81, 88, 95, 98, 100, 104, 106, 108).

There is a group of cases (Nos. 40—45) which at first sight one would be inclined to place together as a fourth class. I refer to those in which hæmoglobin is detected by

the guaiacum test but in which albumen cannot be found in any stage by the ordinary methods of testing. The difference between these cases and those I have grouped above as Class 2 is only apparent, and in every case albumen can be detected by appropriate means. The majority present only an occasional trace of hæmoglobin, and it is only after careful concentration of the urine to a very small bulk that albumen can be demonstrated. Sometimes a trace of hæmoglobin can be detected over a period of several days, but my experience has not furnished me with a single case of true hæmoglobinuria, *i. e.* of a urine with a quantity of hæmoglobin without any blood-corpuscles, although in one or two cases a deceptive resemblance to this was caused by the presence of a small quantity of blood in a highly-coloured urine.

Dropsy without Albuminuria.

It is well known that some curious cases of scarlet fever occur, in which œdema of certain parts of the body is found, while no evidence of kidney mischief can be detected on examining the urine. Of such cases I have seen only two in which the swelling was at all well marked. One of these was a boy, four years of age, who was admitted to the hospital with measles. From this he was making a good recovery, when he was attacked with scarlet fever of a most malignant type, from which he died after an illness of only a few days. Two days before death the face, limbs, and trunk, presented very considerable swelling. Not a trace of albumen or blood was found in the urine, although these were very carefully and frequently tested for. The urine was scanty, high coloured, turbid and loaded with urates. There was no post-mortem examination. The second case presented very decided swelling of the face and legs, commencing on the ninth day, and lasting for from five to six days; yet the most careful testing of the urine failed to reveal the minutest trace of

albumen or blood. The patient made a good recovery, and in fact this complication seemed to cause no inconvenience whatever. Although these are the only two cases I have seen in which there could be no doubt about the existence of œdema without albuminuria, I am inclined to believe that slighter cases of the same kind are not uncommon. I have frequently seen, or perhaps I should say suspected, puffiness of the face during convalescence from scarlatina, but so slight that two observers might probably have differed about its presence. In these cases there was, of course, no albuminuria to assist in coming to a conclusion on this point.

Leaving out of sight the first case quoted, in which the alteration in the constitution of the blood, caused by an overwhelming dose of scarlatinal poison, might have been the cause of death, almost all such cases seem to make a good recovery,[1] *i. e.* the attack of nephritis (if the œdema be due to this) is very slight. Everyone who makes a practice of examining the urine of scarlatinal patients, even once a day, is familiar with the fact that now and then the detection of albumen in the urine is preceded, often for a day or more, by the occurrence of œdema,—of the face more particularly. If at this point the nephritis become arrested we have a case of "dropsy without albuminuria." Nephritis without albuminuria is an uncommon condition, yet one of the existence of which there can be no doubt, and it would seem very reasonable to look upon cases of dropsy without albuminuria as simply slight cases of nephritis which have rapidly resolved, just as occurs in so many cases where the nephritis is characterised by mere traces of albumen and no dropsy. This is the more probable since we are aware that albuminuria is by no means the earliest sign of nephritis, the first rise in arterial pressure revealed by the sphygmograph preceding it, in some cases, often by a considerable interval. It is very probable that the vessels of some individuals are predisposed to permit exudation of their contained fluids into the cellular

[1] 'Niemeyer's Practical Medicine,' Art. "Scarlatina."

tissue on the slightest irritation by the uræmic poison, and it may be in some such manner as this that dropsy without albuminuria is produced.

IV. It will be convenient at this point to discuss the phenomena of the so-called "PRE-ALBUMINURIC STAGE." By this term I understand that what is usually meant is a stage in nephritis characterised by increased vascular tension and, as a result, the presence of blood crystalloids in the urine *before* albumen makes its appearance. The present investigations would lead me to the opinion that such a stage does not really exist, in so far at least, as the absence of albumen in the earliest stages of the nephritis is concerned. I greatly regret the loss of a number of pulse tracings which I made and of which I am unable to give copies; what was observed would lead me to agree with those who maintain the existence of a very early stage in this affection, characterised by the arterial pressure rising steadily for a period of twenty-four hours or more before *anything abnormal* can be discovered by an examination of the *urine*. I cannot therefore see my way to recognise the existence of a "pre-albuminuric stage" characterised by a rise in the blood pressure, that rise in pressure being accompanied or followed by the presence of hæmoglobin in the urine before albumen can be detected. As my table at the end of the paper shows, only ten of all the cases of nephritis observed had a "pre-albuminuric stage" within the latter meaning, whereas most cases I have observed present a rise in blood pressure before albumen or blood appears. In short, there is a "pre-albuminuric stage" in which the blood pressure rises, and this seems to exist indifferently, whether the case subsequently becomes one of albuminuria pure and simple or one of hæmaturia, and this even when the attack is mild. This fact alone is, I think, quite sufficient to lead us to reject the theory that albuminuria in its earliest stage is to be accounted for by the increase in blood pressure alone, and that this stage is characterised by the presence of blood crystalloids. It seems to me much more reasonable to look upon the rise

in the blood pressure as a secondary phenomenon, perhaps due to inefficient innervation of the vascular system, and to regard the extravasations found in the tissues of scarlatinal patients as a result of degeneration of the capillaries and smaller vessels. As above mentioned, only ten of all the cases of nephritis examined showed traces of hæmoglobin *before* albumen could be detected by the ordinary methods. I say by the ordinary methods, for that albumen is present in the urine along with the first traces of hæmoglobin I shall now endeavour to show. If the urine of the so-called "pre-albuminuric stage" of Mahomed[1] be rapidly evaporated in a current of cold, dry air, then filtered and tested, 1st with nitric acid in the cold, 2nd by boiling, and 3rd with picric acid as previously described, in almost all cases the characteristic reaction of albumen will be obtained. In one or two cases where nitric acid failed, after evaporation, to give the usual ring, the presence of albumen was indicated by the boiling and picric acid tests. In one or two cases, picric acid indicated a trace of albumen, while nitric acid and boiling failed to demonstrate its presence even after concentration. In these cases, however, the quantity of urine available for examination was limited, and I am confident that if the evaporation had been carried further the urine would have given characteristic reactions with all three tests. I am of opinion that if a test could be found for albumen as delicate as guaiacum is for blood, the former substance would be invariably detected in the urine of the "pre-albuminuric stage," without any concentration. This opinion is further justified by the existence of what might be called a "post-albuminuric stage." This condition was found in twenty of the patients examined. In these cases traces of blood-colouring matter were detected in the urine, long after all traces of albumen had ceased to be detected by ordinary means. In one or two cases this stage extended over a period of nearly two months, the quantity of hæmoglobin varying from time to time; but it was always noticed that

[1] "Ætiology of Bright's Disease," 'Medico-Chirurg. Trans.,' vol. lvii.

when the quantity of hæmoglobin increased beyond a trace, albumen put in an appearance with the ordinary tests, thus indicating that it had probably been there all along. This stage I regard as entirely analogous to the "pre-albuminuric stage." The apparent absence of albumen in the "pre-albuminuric" and "post-albuminuric" stage is paralleled by what is often seen on examining the urine of menstruating women and by what one finds on direct experiment. From observations conducted upon a number of women whose urine was tested several times daily with great regularity, it was found that in some of the cases, at the menstrual periods, the guaiacum test revealed the presence of blood before nitric acid indicated the presence of albumen. The same peculiarity was observed as the menstrual flow was passing off. There can be no doubt that in the urine of these women albumen as well as hæmoglobin was present, the blood being altered in some of its properties, yet containing these two constituents. It cannot be doubted, I think, that the urine contained blood pure and simple, and yet only hæmoglobin could be detected by the guaiacum test, while nitric acid failed to give any reaction at all. On concentration of the urine albumen was found. The same conclusion is proved by the following experiment: If a drop of fluid blood be placed in a conical glass and normal urine gradually added, as dilution goes on albumen will be found to cease to give a reaction with nitric acid some time before the guaiacum test ceases to react with the hæmoglobin, it being understood that the mixture is allowed to rest after each dilution and that the urine to be tested for hæmoglobin is taken from the bottom of the glass. This early apparent disappearance of the albumen is what one would naturally expect, even if the nitric acid and guaiacum tests were equally delicate; for, while the albumen is dissolved and diffused throughout the fluid, the corpuscles containing the colouring matter (hæmoglobin) sink to the bottom, only a small quantity of the hæmoglobin being dissolved out by the urine. To my thinking, the facts noted above

are pretty strong evidence in favour of the existence of traces of blood pure and simple in the so-called "pre-albuminuric" and "post-albuminuric" stages, even if the presence of albumen had not been demonstrated by the method of concentration.

The next point of inquiry is as to the sediments present in the urine of the "pre-albuminuric stage." The sediment of urine passed during this stage contains both blood-corpuscles and tube-casts. In the first place the presence of corpuscles is to be expected where we have both albumen and hæmoglobin. The actual presence of corpuscles, however, is not so easy to determine by the microscope, and this is not to be wondered at when we remember that a very considerable quantity of urine of the "pre-albuminuric stage" is necessary sometimes to give the reaction with guaiacum in spite of the great delicacy of that test. It is often trying to one's patience to have to search through two or three drachms of urine, drop by drop, for corpuscles, and the difficulty is increased by the fact that if the urine be allowed to settle for too long a period, the corpuscles become altered, sometimes almost beyond recognition; yet even in these cases I have usually found a patient search rewarded by the discovery of red corpuscles, in sufficient numbers to account for the sediment reacting with guaiacum, without having to assume the presence of dissolved hæmoglobin. If such urine be repeatedly filtered through a thick layer of cotton wool and then allowed to settle, it will be found that the urine from the bottom of the glass has ceased to react with guaiacum, while the cotton wool used as the filtering medium gives the characteristic reaction, *i. e.* the cotton has separated the solid corpuscles from the fluid portion of the urine.

The following experiments indicate that the colouring matter is chiefly contained in the first instance within some protective covering, such as a cell wall or protoplasmic mass, and is only slightly in solution shortly after the urine has been passed. If urine from a case such as

we are now considering be put into a test-tube and a little of it examined, the same quantity of hæmoglobin will be found at whatever depth the urine may be taken from. If the tube be now allowed to stand for some time and the urine be again tested, the examination being conducted at different levels, it will be found that the upper layers give a less decided reaction than the lower, and that the depth of the blue colour increases as we approach the bottom, the quantities of urine and reagents being the same in each experiment. This would seem to indicate that the colouring matter is solid or of greater specific gravity than the fluid. If now the tube be shaken up every hour for a period of ten or twelve hours, and then be allowed to settle over night, it will be found that the upper layers give a reaction with guaiacum which is much more decided than that obtained with the same reagent after the urine has merely been allowed to stand for the same length of time. This seems to show that corpuscles contain the colouring matter, that these first of all settle gradually towards the bottom of the vessel, and that after a time a great part of the hæmoglobin is dissolved out, and diffuses itself throughout the fluid.

Of the many sediments besides blood-corpuscles found in the urine of scarlatinal patients, we are interested mainly in tube-casts. These I observed only three or four times in the urine passed in the "pre-albuminuric stage." They were mostly epithelial in character, and were noticed usually only a few hours before the time at which albumen was first detected. In one case tube-casts (epithelial and blood) were found very abundant in the urine six days before the detection of albumen by the usual methods. During this period guaiacum indicated the presence of blood, and white and red corpuscles were detected microscopically. In this case there was no history of previous kidney mischief.

V. Treatment.

To this I shall refer very briefly. I have not been able to satisfy myself that the action of purgatives is really specific in preventing the occurrence of albuminuria. Almost every case admitted to my wards had castor oil administered every third day, so that the bowels were kept moderately free, and yet albuminuria occurred in a large proportion of the cases. Some of these were very severe, and in a few death resulted. One may be misled in regard to the efficacy of purgatives by the occurrence of what is not uncommon in scarlet fever, viz. the appearance of blood or albumen for perhaps only a few hours, which disappears without any treatment whatever. If purgatives have been used in such cases one would be apt to refer to the action of the medicine what is really part of the natural course of the disease.

Warmth and rest seem, after all, the most efficient guards against albuminuria, although these frequently fail in their object.[1]

I may mention here that I was in the habit of confining my patients to bed during the first four weeks of the fever, and that they were not allowed to leave the ward till a week later. By confining the diet to milk and farinacea during the first two or three weeks of the scarlatina, and allowing beef broths, &c., only when convalescence began to be established, I attempted to ward off nephritis. In thirty cases milk and farinacea were continued till the middle of the fifth week, yet nine of these cases showed signs of albuminuria; in most cases these were slight, one only being a well-marked case of scarlatinal dropsy. Whether this diminished percentage of albuminuria was due to the mild nature of the diet, or to accident, all the cases having occurred in early autumn,

[1] The temperature of the wards, built on the pavilion system with efficient through and roof ventilation, was maintained as near 60° Fahr. as possible.

I cannot say. The converse of this experiment I did not care to try.

After albuminuria has attacked a patient the usual treatment with purgatives and packs seems very effective in most cases.

Convulsions are best combated by chloral and chloroform, but these agents can check only the more urgent symptoms and afford time for more routine remedies to act. Benzoic acid in large doses (twenty grains every two hours) seemed to have a powerful influence, at least in some cases, in preventing the occurrence of convulsions.

In recapitulation I would recall the following points :

I. All cases of scarlatinal albuminuria may be subdivided into :

(*a*) "Initial" albuminuria.

(*b*) "Late" albuminuria.

This distinction is to some extent arbitrary, but the actual conditions found in many cases seem to justify it.

II. All cases may be subdivided into three classes :

(*a*) Cases of simple albuminuria.

(*b*) Cases of simple hæmaturia.

(*c*) Cases in which there are both blood and albumen, but in which albumen is in excess.

III. There is no condition of the urine which justifies the use of such a phrase as "pre-albuminuric stage." If such a term is to be used at all it should refer to the condition of the vascular system only.

IV. Lastly, red and white corpuscles and tube-casts are commonly found in the urine during the so-called "pre-albuminuric stage."

(For a report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' New Series, vol. ii, p. 11.)

TABLE

Giving details of Observations made upon the Urine of 112 *Cases of Scarlatinal Nephritis.*

TABLE GIVING DETAILS OF OBSERVATIONS MADE UP

Min. tr. = minute trace; tr. = trace; dist. = distinct; con. = considerable; abdt. from one date to another; (a.m.) or (p.m.) added to a date indicates that the albun otherwise it was pres

A. *Cases of "Initial Albuminuria*

No. of case.	Date of admission.	Age.	Sex.	Day of illness.		Periods at which albumen was detected. Number day of illness. Abbreviations as above.
				Adm.	Dism.	
1	Dec. 15	22	F.	2nd	54th	4th tr., 5th abdt., 6th—10th tr., 41st tr.
2	Dec. 26	7	F.	5th	90th	8th (a.m.)—70th varying from tr. to con.
3	Jan. 23	6	F.	3rd	60th	5th (p.m.)—14th (a.m.) tr.—dist.
4	Jan. 30	3	F.	7th	64th	8th (a.m.) min. tr., 9th (p.m.) dist. 10th, 11th (36th (a.m.)—38th (a.m.) tr., 58th (p.m.) tr.
5	Feb. 3	11	F.	5th	59th	5th—10th con., 35th (p.m.)—41st (a.m.) di 50th, 52nd tr.
6	Feb. 7	5	F.	2nd	54th	4th (a.m.)—32nd (p.m.) varying from tr.—con.
7	Jan. 23	4	F.	1st	57th	7th (p.m.), 9th (a.m.) dist., 10th (a.m.)—14th (p. 21st (p.m.), 24th (p.m.), 27th (a.m.), 32nd (p.r 33rd (p.m.), 34th (p.m.), 36th, 37th, 39th (p.r 40th (p.m.) tr.
8	Apr. 5	4	F.	2nd	81st	3rd (p.m.), 4th (p.m.), 8th (p.m.), 11th (a.m.), 1 (p.m.), 18th (p.m.) tr.
9	Apr. 16	6	M.	3rd	16th	4th—8th (p.m.) tr., 8th (p.m.)—13th dist.

B. *Cases of "Initial Albuminuria" followed*

10	Aug. 17	6	M.	5th	54th	5th abdt., 9th (p.m.)—29th tr.—abdt.
11	Nov. 10	3	F.	3rd	24th	8th tr., 18th—21st con., 22nd—24th tr.
12	Dec. 12	10	M.	6th	60th	7th (a.m.) tr., 15th (a.m.) dist., 39th (p.m.) tr.
13	Jan. 23	30	F.	2nd	56th	6th (a.m.), 10th (a.m.) tr.
14	Jan. 23	6	F.	3rd	66th	3rd—7th con., 16th (a.m.) tr., 18th (a.m.) (22nd tr., 25th, 26th (a.m.) tr., 34th (a.m.) 48th (p.m.) min. tr.—dist.
15	Jan. 23	8	F.	3rd	66th	4th—9th (p.m.) tr., 12th (a.m.), 13th (a.m.), 1 (p.m.), 18th (p.m.), 21st (p.m.) tr., 23rd (a.r —51st (p.m.) tr.
16	Feb. 21	22	M.	2nd	84th	3rd, 4th, 5th dist., 22nd—38th tr.—dist.
17	Mar. 2	6	M.	5th	57th	6th—8th dist., 12th (p.m.) dist.
18	Mar. 3	19	F.	6th	55th	6th, 7th tr., 15th (p.m.) con., 27th tr., 28th tr.
19	Mar. 15	4	M.	3rd	60th	4th (p.m.), 10th (a.m.) min. tr., 13th—19th mi tr.—dist.
20	Mar. 26	7	M.	21st	94th	3rd—7th tr., 11th (a.m.), 13th (a.m.) min. t 22nd (a.m.)—24th dist.

HE URINE OF 112 CASES OF SCARLATINAL NEPHRITIS.

bundant; oc. = occasional; in. = initial. A dash — indicates continuance of the albumen as only found in the morning or evening sample of that day as the case may be; all three testings.

unning on to "Late Albuminuria."

Hæmoglobin detected, number day of illness.	Duration of nephritis.	"Pre-albuminuric stage."	"Post-albuminuric stage."	Dropsy.	Result.	Remarks.
5th dist.	5 days	None	None	Con.	Well	
None	62 days	None	None	Con.	Well	
None	9 days	None	None	None	Well	
58th (p.m.) tr.	4 days oc. tr.	None	None	None	Well	
None	5 days and oc. tr.	None	None	None	Well	
None	28 days	None	None	None	Well	
3rd (a.m.) min. tr., 33rd (p.m.), 36th (p.m.) dist., 37th (p.m.), 39th (p.m.), 40th (p.m.) tr.	8 days and oc. tr.	None	None	None	Well	
None	Oc. tr.	None	None	None	Well	
None	9 days (?)	None	None	None	Died	

Late Albuminuria" after a varying interval.

th dist., 9th—24th tr.—dist.	Date 20 days	None	None	Dist.	Well	
None	7 days	None	None	None	Died	
44th (a.m.) tr.	Oc. tr.	None	None	None	Well	
None	Tr. on 2 oc.	None	None	None	Well	
None	32 days Init. 5 days	None	None	None	Well	
46th (p.m.)—48th (p.m.) tr.	41 days, in. 5 days	None	None	Con.	Well	
2nd—70th tr.—dist.	58 days, in. 3 days	None	52 days	None	Well	
12th (p.m.) con.	3 days	None	None	None	Well	
None	In. 2 days, oc. tr.	None	—	None	Well	
None	7 days and oc. tr.	None	None	None	Well	
Dist.—tr. from 3rd—45th, 50th min. tr.	43 days	None	21 days	None	Well	Long "post-albuminuric stage." Long - continued presence of hæmoglobin and occasional alb.

No. of case.	Date of admission.	Age.	Sex.	Day of illness.		Periods at which albumen was detected. Number day of illness. Abbreviations as above.
				Adm.	Dism.	
21	April 5	22	F.	2nd	57th	3rd—6th dist., 9th (a.m.) min. tr., 22nd—24 dist.
22	April 17	19	M.	2nd	150th	2nd—6th tr., 31st (a.m.) min. tr., 56th min. t 57th dist., 58th—63rd dist.
23	May 2	6	F.	1st	54th	7th (a.m.), 8th (p.m.) tr., 11th (p.m.)—15th (p.m tr., 17th (p.m.) tr., 18th (p.m.) tr., 36th (a.m —46th (p.m.) tr.—dist.
24	May 6	6	F.	6th	26th	6th and 7th tr., 15th (p.m.)—20th (a.m.) tr.—co
25	May 11	11	F.	3rd	57th	3rd tr., 23rd (a.m.) dist., 28th (p.m.), 29th (p.m dist., 34th (p.m.) con., 36th (a.m.) min. tr., 37 (p.m.)—53rd (p.m.) tr.—dist.
26	June 30	15	M.	1st	?	1st, 2nd dist., 3rd min. tr., 5th (a.m.) min. t 19th—84th dist.—abdt.
27	July 2	35	M.	5th	56th	5th—9th tr.—dist., 21st (p.m.) tr., 23rd (p.m.) t
28	July 2	26	M.	4th	66th	4th (p.m.) tr., 19th (p.m.)—40th (p.m.) con.—abd 41st—52nd tr., 64th (p.m.) min. tr.
29	Aug. 22	18	F.	5th	?	5th (p.m.)—9th (a.m.) tr., 14th, 15th tr., 16th 41st tr.—dist.
30	Feb. 1	7	M.	21st	94th	3rd—7th tr., 11th (a.m.), 13th (a.m.) min. tr., 22 (a.m.), 24th (a.m.) dist.

C. *Cases of "Initial Albuminuria*

31	Dec. 26	16	F.	?	?	7th (p.m.) dist., 8th (a.m.) and (p.m.) tr.
32	Jan. 29	22	F.	3rd	62nd	4th—6th (p.m.) dist., 7th (a.m.) tr.
33	Feb. 21	27	M.	4th	54th	4th—8th dist.
34	April 25	7	M.	3rd	55th	8th (a.m.) min. tr.
35	June 7	35	F.	6th	57th	7th (a.m.) tr.
36	June 28	27	M.	4th	55th	4th con., 5th tr.
37	July 2	13	F.	5th	56th	7th—9th tr.
38	July 4	26	F.	2nd	56th	2nd—5th tr.—con.
39	Aug. 14	3½	M.	5th	56th	7th (p.m.) tr., 8th (a.m.) tr.
40	Aug. 28	28	F.	2nd	55th	2nd con., 3rd dist., 4th (a.m.) tr.

D. *Cases of Hæmoglobin*

41	Dec. 9	2	F.	10th	65th	None
42	Feb. 21	9	M.	10th	62nd	None
43	March 1	62	F.	14th	56th	None
44	April 24	6	F.	3rd	53rd	None
45	June 21	8	M.	7th	62nd	None
46	June 21	11	F.	5th	59th	None

Hæmoglobin detected, number day of illness.	Duration of nephritis.	"Pre-albuminuric stage."	"Post-albuminuric stage."	Dropsy.	Result.	Remarks.
7th (a.m.) tr.	4 days, in. 4 days	None	None	None	Well	
2nd—3rd dist., 3rd (p.m.)—57th (a.m.) tr., 57th—65th (p.m.), dist., 66th—106th tr.	103 days	None	43 days	Con.	Well	
None	10 days and oc. tr.	None	None	None	Well	
None	5 days, in. 22 days	None	None	None	Died	Malignant.
None	20 days and oc. tr.	None	None	None	Well	
19th—65th min. tr.—abdt.	61 days, in. 5 days	None	None	Con.	Well	
21st (p.m.)—24th (p.m.) tr.	4 days	None	24 hours	None	Well	
19th—66th (p.m.) tr.—con.	47 days	None	14 days	None	Well	Case sent to country.
5th tr., 8th tr., 14—20th tr.—dist.	36 days	None	None	None	Well	
3rd—45th dist.—tr., 50th min. tr.	43 days	None	25 days	Slight	Well	Long "post-albuminuric stage."

not followed by "Late Albuminuria."

None	2—3 days	None	None	Dist.	Died	Malignant.
None	4 days	None	None	None	Well	
None	5 days	None	None	None	Well	
None	—	None	None	None	Well	
None	—	None	None	None	Well	
None	2 days	None	None	None	Well	
None	3 days	None	None	None	Well	
None	4 days	None	None	None	Well	
None	2 days	None	None	None	Well	
None	3 days	None	None	None	Well	

Urine without obvious Albumen.

26th tr.—dist.	—	None	None	None	Well	No albumen detected till urine concentrated.
13th tr.	—	None	None	None	Well	
17th tr.	—	None	None	None	Well	
17th min. tr., 18th (a.m.) dist., 18th (p.m.) min. tr.	40 hours	None	None	None	Well	
2nd—4th (a.m.), min. tr.	—	None	None	None	Well	
21st (a.m.) tr.	—	None	None	None	Well	

B. *Cases not*

No. of case.	Date of admission.	Age.	Sex.	Day of illness.		Periods at which albumen was detected. Number day of illness. Abbreviations as above.
				Adm.	Dism.	
47	Nov. 10	9	F.	4th	61st	35th, 37th, 40th, 42nd, 47th, 49th, 55th (a.m.) tr., 51st, 52nd, 53rd, 55th (p.m.), 56th, 59th, 61st con.
48	Nov. 13	10	F.	11th	62nd	32nd—39th tr.
49	Nov. 17	7	F.	1st	57th	39th tr.
50	Nov. 17	7	M.	4th	57th	12th—16th, 20th, 22nd, 23rd, 24th, 27th, 30th tr., on other days from 12th till 30th dist.
51	Nov. 22	12	M.	3rd	56th	17th, 23rd, 24th min. tr.
52	Nov. 23	6	M.	7th	56th	14th tr.
53	Nov. 28	9	F.	7th	56th	19th (p.m.) and 31st (a.m.) tr.
54	Nov. 28	8	F.	7th	59th	29th (p.m.), 35th (a.m.), 42nd (p.m.), 43rd (a.m.) con., 46th tr., 50th (p.m.), 53rd (p.m.) tr.
55	Nov. 29	22	M.	3rd	76th	32nd (p.m.) tr., 33rd abdt., 34th—38th vary., 36th tr., 38th till 42nd vary., 47th—76th vary.
56	Dec. 2	11	M.	2nd	142nd	16th (p.m.) tr., then abdt. till 77, then oc. till 101st
57	Dec. 2	23	M.	7th	162nd	20th tr., 21st—48th abdt., 49th—57th dist., 58th—64th con., 65th—84th dist., 85th—162nd tr.
58	Dec. 6	12	M.	4th	54th	37th (a.m.), 40th, 41st, 43rd (a.m.) tr.
59	Dec. 6	11	F.	6th	58th	16th (p.m.) dist.
60	Dec. 7	3	M.	6th	17th	15th (a.m.) tr., 15th (p.m.) till end con.
61	Dec. 7	5	M.	4th	56th	36th (p.m.) dist., 40th (a.m.) dist., 42nd (a.m.) tr.
62	Dec. 8	10	M.	4th	82nd	36th (p.m.)—73rd (p.m.) tr. to dist.
63	Dec. 8	5	M.	5th	75th	35th (a.m.), 49th, 52nd, 54th, 58, 59th (p.m.) dist.
64	Dec. 10	20	F.	4th	55th	15th (a.m.) tr., 34th (p.m.) tr., 35th (p.m.) dist., 37th (p.m.) dist., 38th (a.m.) dist., 46th (p.m.) dist.
65	Dec. 12	3	F.	14th	68th	15th (a.m.) tr., 22nd (p.m.) con., 23rd (a.m.) tr., 37th (a.m.) dist., 39th (p.m.) tr., 45th (a.m.) tr., 52 (p.m.) tr.
66	Dec. 12	6	M.	Weeks 1½	31st	16th till end abdt.

strictly classified.

Hæmoglobin detected, number day of illness.	Duration of nephritis.	"Pre-albuminuric stage."	"Post-albuminuric stage."	Dropsy.	Result.	Remarks.
None	26 days	None	None	None	Well	Albumen occurred only 8 p.m., except when noted.
None	2 oc. tr.	None	None	None	Well	Traces morning.
None	Once tr.	None	None	None	Well	
27th, 28th, 29th, 30th, 31st tr., 35th tr.	19 days	None	1 day on 35 tr.	None	Well	Albumen all at night.
26th tr.	Thrice tr.	None	None	None	Well	Albumen all at night.
None	Once tr.	None	None	None	Well	
None	Twice tr.	None	None	None	Well	
None	Occasionally	None	None	None	Well	
None	44 days	None	None	None	Well	
15th tr. and dist., 16th till 77th abdt. and con., 77th—139th tr.	122 days	36 hours	36 days	Con.	Well	During "post-albuminuric stage" minute traces of albumen were observed occasionally. Duration of "post-albuminuric stage" uncertain, patient being dismissed with trace of blood.
19th (a.m.) — 21st (p.m.) tr., 22nd (a.m.).—44th abdt., 45th — 75th dist., 76th—162nd tr.	142 days	24 hours	24 hours dismissed with tr. of blood	Con.	—	
None	Oc. tr.	None	None	Slight 25th day	Well	
16th (p.m.) dist., 17th (a.m.) tr.	Once tr.	None	12 hours	None	Well	
None	3 days	None	None	Abdt.	Died	Malignant.
19th, 20th dist.	Oc. tr.	None	None	None	Well	Blood and albumen to usual tests appeared at different times.
16th (p.m.) tr., 36th (p.m.)—51st (p.m.) min. tr.—dist.	37 days	None	None	Slight 35th, 41st	Well	Except on 35th albumen always at night.
15th (p.m.) dist., 16th (a.m.) tr.	Oc. tr.	None	None	None	Well	
13th (p.m.) — 15th (a.m. and p.m.) and 16th (a.m.) tr.	Oc. tr.	None	None	None	Well	
19th (p.m.) — 23rd (a.m.) dist., 23rd (p.m.) tr., 35th (a.m.) tr., 37th (a.m.) tr.	112 hours and oc. tr.	72 hours	16 hours	None	Well	
15th tr., 16th till end cons.	?	1 day	?	Con.	Died	

No. of case.	Date of admission.	Age.	Sex.	Day of illness.		Periods at which albumen was detected. Number day of illness. Abbreviations as above.
				Adm.	Dism.	
67	Dec. 12	10	M.	3rd	71st	9th (p.m.)—44th (a.m.) vary. from min. tr.—cons.
68	Dec. 13	4	F.	7th	75th	17th (p.m.) tr., 45th (p.m.) dist., 56th (p.m.) tr.
69	Dec. 14	4	F.	4th	65th	15th (p.m.) tr., 24th (a.m.) dist., 52nd (a.m.) con., 53rd (p.m.) dist., 56th (p.m.) tr.
70	Dec. 14	7	M.	3rd	20th	12th, 13th (p.m.) tr., 13th, 14th (a.m.) dist., 14th (p.m.)—end, abdt.
71	Dec. 14	14	M.	4th	57th	35th (p.m.), 39th (a.m.) tr.
72	Dec. 14	13	F.	8th	82nd	15th—30th dist.—cons., 35th—44th (p.m.) tr., 47th—52nd tr., 59th, 61st tr., 68th—70th min. tr.
73	Dec. 15	8	F.	1st	53rd	23rd (p.m.) tr.
74	Dec. 17	8	M.	2nd	55th	25th (a.m.) tr.
75	Dec. 27	8	M.	2nd	72nd	15th (a.m.) min. tr., 21st (a.m.) tr., 28th (a.m.) tr., 35th (a.m.) tr., 37th (a.m.) tr., 38th (a.m.) tr., 47th (a.m.) tr.
76	Dec. 27	6	M.	8th	50th	10th (a.m.) tr., 16th (a.m.) tr., 21st, 27th (p.m.) tr., 35th (a.m.) tr., 39th (p.m.) tr., 44th—49th dist.
77	Dec. 26	21	F.	4th	56th	32nd tr.
78	Dec. 29	7	F.	4th	68th	15th (p.m.)—33rd (a.m.) vary.from cons.—min. tr., 41st (a.m.) dist., 52nd (p.m.) min. tr.
79	Dec. 30	10	M.	3rd	58th	9th (a.m.), 10th (p.m.) min. tr.
80	Feb. 3	4	F.	8th	59th	12th (a.m.), 19th (a.m.) tr.
81	Feb. 10	6	F.	4th	153rd	11—109th very vary. from abdt.—min. tr.
82	Feb. 7	9	F.	2nd	64th	27th tr.
83	April 2	7	F.	14th	122nd	22nd (p.m.)—35th (a.m.) con., 35th (p.m.) tr., 39th dist., 40th—43rd (p.m.) tr.
84	April 14	7	F.	2nd	60th	47th (p.m.), 52nd (p.m.) tr.
85	April 23	8	F.	2nd	55th	46th (p.m.) tr., 51st (a.m.) tr.
86	April 25	4	M.	2nd	63rd	12th (a.m.) dist., 12th (p.m.)—17th (p.m.) abdt., 18th (a.m.), 19th (p.m.) dist., 20th (a.m.) tr.
87	April 28	5	F.	3rd	57th	40th (p.m.) dist., 46th (a.m.) min. tr.
88	May 2	14	F.	3rd	55th	15th (p.m.) tr., 41st (a.m.)—53rd (p.m.) tr.—cons.

Hæmoglobin detected, number day of illness.	Duration of nephritis.	"Pre-albuminuric stage."	"Post-albuminuric stage."	Dropsy.	Result.	Remarks.
None	35 days	None	None	Slight	Well	Albumen usually most abdt. in m.
None	Oc. tr.	None	None	None	Well	
None	Oc. tr.	None	None	None	Well	
17th (p.m.)—end dist.	8 days ?	None	None	Slight	Died	Uræmia (death).
14th (p.m.), 34th (p.m.), 48th (a.m.) min. tr.	Oc. tr.	None	None	None	Well	Occasional trace of albumen and blood.
15th—50th tr.	56 days	None	None	None	Well	
None	Once tr.	None	None	None	Well	
22nd (p.m.) tr.	Once tr.	None	None	None	Well	
None	Frequent tr.	None	None	None	Well	Albumen, when present, always in morning.
None	Frequent tr.	None	None	None	Died	Times at which albumen appeared very various.
31st—33rd tr.	3 days	1 day	1 day	None	Well	Note continued presence of blood.
15th (p.m.) — 43rd (p.m.) dist. — tr., 52nd (p.m.) — 56th tr.	28 days	None	10 days	None	Well	
7th (p.m.) tr.	Twice tr.	None	None	None	Well	
None	Twice tr.	None	None	None	Well	
17th—135th very vary., abdt. to min. tr.	124 days	None	26 days	Slight	Well	
None	Once tr.	None	None	None	Well	
21st (a.m.) and (p.m.), 22nd (a.m.) tr., 22nd (p.m.), 30th (p.m.) con., 31st (a.m.)—36th (a.m.) tr., 39th (a.m.)—47th (a.m.), tr.	25 days	24 hours	4 days	Dist.	Well	
None	Twice tr.	None	None	None	Well	
None	Twice tr.	None	None	None	Well	
11th tr., 12th dist., 13th (a.m.), 17th (p.m.) cons., 18th (a.m.)—20th (a.m.) dist., 20th (p.m.)—21st (a.m.) tr.	10 days	24 hours	16 hours	None	Well	
None	Twice tr.	None	None	None	Well	
15th (p.m.) dist., 20th (p.m.), 21st (a.m.) min. tr., 21st (p.m.) dist.	12 days and oc. tr.	None	None	None	Well	

No. of case.	Date of admission.	Age.	Sex.	Day of illness.		Periods at which albumen was detected. Number day of illness. Abbreviations as above.
				Adm.	Dism.	
89	May 14	14	M.	8th	47th	31st (a.m.), dist.
90	June 2	18	M.	3rd	169th	21st (a.m.), 27th (p.m.) tr., 28th (a.m.), 34th (p.m.) cons., 35th (a.m.)—58th (a.m.) tr.—dist., 85th (p.m.) tr.
91	June 9	14	M.	7th ?	10th ?	6th—10th dist.
92	June 19	8	F.	10th	18th	10th—18th abdt.
93	June 21	8	M.	10th	115th	10th—50th tr., except 16th, 17th dist.
94	June 21	4	F.	4th	58th	16th (a.m.)—27th (a.m.) tr.—con.
95	June 25	6	F.	6th	140th	16th tr., 17th (a.m.)—68th con.—abdt., 69th—75th dist.
96	June 26	10	M.	3rd	78th	30th (p.m.), 33rd (p.m.) min. tr.
97	June 27	7	F.	2nd	56th	42nd (a.m.) tr.
98	June 30	8	M.	3rd	59th	25th (p.m.) vary. from min. tr., 30th (a.m.), 32nd (p.m.) min. tr.
99	July 3	7	M.	14th	56th	14th, 15th, 16th tr., 18th (p.m.) min. tr., 24th—32nd tr.—con.
100	Aug. 14	3½	M.	10th	88th	15th—53rd tr.—abdt.
101	July 3	6	M.	5th	61st	14th (p.m.), 27th abdt.—tr.
102	July 21	6	M.	4th	23rd	22nd (p.m.) tr., 23rd con.
103	July 25	6	M.	21st	88th	15th—30th tr.—con.
104	Aug. 7	8	M.	7th	109th	17th (p.m.)—54th tr.—abdt.
105	Aug. 13	7	F.	9th	33rd	9th (p.m.) con., 10th (a.m.) dist., 11th—18th tr., 18th—24th con., 24th—33rd abdt.
106	Aug. 13	5	M.	10th	58th	10th—20th (p.m.) tr.—dist.
107	Aug. 17	15	M.	4th	55th	18th (a.m.), 19th (p.m.) tr.
108	Aug. 22	8	M.	10th	60th	14th—16th tr., 16th—34th tr.—con.
109	Aug. 22	9	F.	4th	54th	14th—22nd tr.—dist.
110	Aug. 28	10	F.	7th	55th	18th tr.
111	Aug. 28	?	F.	10th	53rd	17th (p.m.)—33rd tr.—con.
112	Aug. 28	5	F.	5th	56th	9th tr.

Hæmoglobin detected, number day of illness.	Duration of nephritis.	"Pre-albuminuric stage."	"Post-albuminuric stage."	Dropsy.	Result.	Remarks.
None	Once tr.	None	None	None	Well	
16th (a.m.), 31st (p.m.) tr., 32nd (a.m.)—52nd (a.m.) dist., 52nd (p.m.)—87th (p.m.) tr.	72 days	5 days	28 days	Con.	Well	
None	5 days	None	None	Abdt.	Died	
10th—18th abdt.	8 days	?	None	None	Died	
None	40 days	None	None	None	Well	Note in this case increase of albumen on 16th day.
15th (p.m.) tr., 16th—28th tr.—dist., 29th—31st (p.m.) min. tr.	16 days	1 day	3 days	None	Well	
16th (a.m.)—41st (p.m.) dist.—con., 42nd, 50th tr., 62nd (a.m.) min. tr.	59 days	None	None	None	Well	
6th (a.m.) min. tr., 15th (p.m.), 29th (p.m.) tr., 30th—33rd dist., 34th (a.m.)—61st (p.m.) min. tr.—tr.	46 days	14 days	27 days	None	Well	
None	Once tr.	None	None	None	Well	
20th—21st (p.m.) tr., 23rd (p.m.), 34th (a.m.) min. tr.—dist.	11 days	None	None	None	Well	
14th—37th tr.—con.	23 days	None	5 days	Con.	Well	
21st—40th tr.—con.	38 ? days	?	None	None	Well	
14th—20th tr.—dist.	13 days	None	None	None	Well	
None	2 days	None	None	None	Died	Malignant.
None	Doubtful	None	None	None	Well	
28th (p.m.)—32nd (p.m.) min. tr.	37 days	None	None	Con.	Well	
9th—19th tr., 20th—21st dist., 22nd—33rd con.	24 days	?	None	None	Died	Note absence of dropsy with abdt. alb.
13th (p.m.)—17th (a.m.) dist., 18th (p.m.) min. tr.	10 days	None	None	None	Well	
None	2 days	None	None	None	Well	
17th—22nd dist., 23rd—25th tr.	20 days	None	None	None	Well	
None	7 days	None	None	None	Well	
None	Once tr.	None	None	None	Well	
None	16 days	None	None	None	Well	
None	Once tr.	None	None	None	Well	

112 cases of albuminuria.
2 cases of dropsy without albuminuria.
66 cases without dropsy or nephritis.

180 total consecutive cases of scarlatina.

ON SOME POINTS

REGARDING THE

DISTRIBUTION OF BACILLUS ANTHRACIS IN THE HUMAN SKIN

IN

MALIGNANT PUSTULE.

BY

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Received May 11th—Read November 24th, 1885.

THE observations which I wish to bring under the notice of the Society are based upon the following case, the notes of which have been condensed as far as possible.

E. G—, æt. 29, by occupation a maker of knife-cleaning machines, was admitted into University College Hospital, on June 7th, 1884. The diagnosis of malignant pustule had been already made by the Resident Medical Officer, Dr. Maudsley, before I was sent for, and I had only to confirm the diagnosis on seeing the patient. The man, though of good physique, looked very ill; his expression was heavy and anxious, the skin of his head and neck looked dusky and greasy; his tongue was coated and his voice was thick. On the left side of the neck, lying upon

the sterno-mastoid muscle about an inch and a half below the ear, there was a large zone of vesicles surrounding a central eschar of dark brownish colour. The latter was hard, dry, and slightly depressed below the level of the belt of vesicles. These ranged in size up to that of a large split pea, and were filled with turbid yellowish or pink serum; they were very tense and hard. Beyond them the skin was much indurated, the whole sore measuring about 3 × 2 inches, the long axis of the oval lying across the neck. There was no great local heat, but much tenderness. Around this focus of disease the whole of the left side of the neck was much swollen, indurated, tense, and shining, the hardness reaching upwards beyond the ear and on to the cheek, downwards over the clavicle and across the middle line both in front and behind. The hardness was peculiar in its distinctness and unlikeness to ordinary œdema. There was considerable difficulty in swallowing and breathing, owing to the swelling having affected the inner surface of the pharynx. The patient's mind was quite clear and he had had no delirium; he seemed, however, worn out from want of sleep and food; there was a tendency to relaxation of the bowels.

He gave the following account of his illness:

On Wednesday, May 28th, 1884, he noticed a pimple on the left side of his neck, which was red and itched a little. On the following Saturday "a small black head" having developed he squeezed out the contents. At this time there was no particular swelling or redness around; but this was noticed two days later, and poultices were applied. On June 5th he became very feverish, and small vesicles appeared at the point of greatest swelling. These soon burst and discharged pale straw-coloured or pink serum. On the 6th, there was increase of difficulty in swallowing, this having been first noticed on the 2nd; the breathing had also become somewhat embarrassed. There were also marked restlessness, insomnia, and headache. Pain was not limited to the affected spot, but was felt all over the body and to a marked extent in the loins. There had

been anorexia and increasing weakness since the fifth day, and on the ninth day he had two rigors, followed by two more on each of the succeeding days.

He lived at St. John's Wood, but worked near the Tower. He had a good deal of handling of horsehair, bristles, and buff leather, but never raw hides. His own impression was that he had contracted the disease at a barber's where he had had his hair cut and had been shaved; the barber also lived near the Tower.

There was no hesitation as to the treatment. Before operating, however, I carefully examined the serum of the vesicles and the blood for bacilli in the usual way, over and over again, but with a negative result. Still there could be little doubt as to the diagnosis. I therefore directed that a large piece of skin, including the whole area of vesiculation and half an inch beyond, should be excised in its whole thickness. The base of the resulting wound was mottled with dark patches, apparently plugged vessels. It was freely treated with the actual cautery and dressed with iodoform.

The morning after the operation the temperature was normal and the patient much better; he made a rapid recovery from this time. Three days after the operation the blood and discharges were examined, but no bacilli were discovered. The patient left hospital on June 24th, with a small healthy wound still open.

Although the clinical history of true anthrax, both in animals and man, has now been written with completeness in this country by Mr. Davies-Colley,[1] and in Germany by Bollinger,[2] some points regarding its minuter pathology still appear to require further study in different cases. Among these may be mentioned, first, the general distribution of the bacilli anthracis in the affected skin round the point of inoculation in man, and next, their relation to the production of the vesicles and eschar so characteristic of

[1] 'Med.-Chir. Trans.,' vol. lxv, 1882.

[2] Ziemssen, 'Handbuch der speciellen Pathologie,' Band iii (Translation, vol. iii).

the disease. In reading the literature of the subject, one is struck with the small amount of attention which these two points appear to have received in this country, indeed, with the exception of Dr. Charlewood Turner's admirable report of the microscopic appearances in Mr. Davies-Colley's case, I am not aware of any native source of information regarding them. The case now recorded offers such a good opportunity of studying the local disease that I have thought it not unworthy the notice of the Society. Generally speaking, it shows a close resemblance to the condition of things described by Dr. Turner. But there are some points regarding the distribution of the bacillus in which the two cases appear to differ, and there are others again a study of which in this case enables us perhaps to carry our observations a little further than Dr. Turner has done.

It is not improbable that the organisms may behave differently in and about the locality of inoculation, in different cases, or may vary in their habits at various stages of the disease. It is only by an accumulation of data bearing upon these questions that we shall be able to explain the very remarkable fact, now firmly established, namely, that free excision of the diseased area around the malignant pustule is followed, in a large proportion of cases, by rapid disappearance of all constitutional disturbance and by complete recovery. This was almost a startling feature in the present case. The disease had reached the eleventh day, the constitution was evidently profoundly affected, there had been several rigors, there were insomnia, anorexia, and great depression lasting for days, besides which the whole side of the neck was in a state of the most intense hardness, and yet after removal of the piece of skin, including the circle of vesicles, immediate disappearance of the constitutional and local symptoms resulted, and the patient was practically well next day.

This is a fact most difficult to explain. Many hypotheses may, of course, be advanced in an effort to clear it up;

but it appears to me that, before everything, we need facts regarding the local habits of the bacilli anthracis in and about the malignant pustule, accumulated from the careful examination of a large number of cases occurring in the human subject. One very significant point is noticeable in this case, and is also alluded to by Dr. Turner, namely, that the bacilli appear to have a strong predilection for the most superficial parts of the skin, and for them only. If this rule should hereafter be shown to hold good in numerous other cases, it will strengthen the hypothesis that the organism can only attain to its fullest degree of virulence in the presence of light and air, and that though it may be carried to deeper parts of the body and perhaps increase there in a measure, nevertheless the original colony around the focus of inoculation on the surface may remain the principal, if not the only, generator of the actual poison, whatever it may be, which depresses the vital powers so powerfully. At present, however, I should prefer to pass by such hypotheses and to range myself with those who are endeavouring simply to accumulate such data as those to which I have just alluded.

The diseased skin immediately after excision was dropped into absolute alcohol and when hardened was frozen, cut, and stained in the usual way. The resulting microscopical sections were particularly satisfactory and from them I made the accompanying drawings (see Plate III) while the colours were vivid and sharply defined.

The first point noticed with the naked eye about the portion of skin excised was a peculiar dark mottling of its under surface corresponding to the area of the malignant pustule (fig. 1). This mottling appeared to be produced by either an intense congestion with some extravasation of blood, or, what seemed equally probable, a thrombosis of vessels with staining around them. The same appearance was noticed on the surface of the wound left by the excision of the skin. The next point noticeable was a distinct swelling of the diseased area, so that the corium was about twice as thick here as elsewhere.

This swelling diminished rapidly at the outer margin of the vesicles. The latter were of the flattened variety and covered an oval area around the central, dark, dry eschar (fig. 1). They were filled with pinkish serum for the most part. Their size was greater towards the advancing margin as if they had dwindled towards the dark, central area. The latter, on section, was drier and tougher than the rest of the skin.

On examination with the microscope, one is first struck with the great abundance of bacilli immediately under the vesicles and their fewness beneath the dry area of the eschar. In the larger vesicles they appear in smaller number than in the more minute, probably owing to their having been, for the most part, washed out in preparation of the sections. In some of the small commencing vesicles, on the other hand, they are packed as closely as possible and form a dark mass filling the space completely.

In the deeper layers of the rete mucosum and at the apices of the papillæ they are more abundant than anywhere else (fig. 2, *b*). Here they are seen by the hundred, packed so closely that under a low power they form a continuous, dark, waving streak following the outline of the papillæ. They are also seen to descend along the root sheaths of the hairs and are there in particularly large numbers (fig. 3, *c*). In contrast to all this, the bodies of the papillæ themselves show so very few bacilli as to suggest that any that are present have only been deposited there in the process of section cutting (fig. 2, *b*, *c*). Again, in the vessels of the papillæ I have not been able to find any organisms, though they have been carefully looked for.

The mode of formation of the younger vesicles is well seen in several of the sections, *e.g.* fig. 2. The irritation of the organisms in the deeper layers of the rete has caused an outpouring of serum among the cells underlying the epidermis, which has gradually forced the latter upwards forming loculi filled with fluid, between which delicate

columns of rete cells may be seen (fig. 2, *a*, *a*). Between these columns or bands of cells the bacilli are aggregated in dense masses in the smaller loculi, but in the larger they are found generally only around the borders, having apparently been washed out from the centre of the space in the process of preparation of the sections. Where no vesicles have yet formed, the apices of the papillæ are seen to swarm with bacilli and appear softened and somewhat broken up in consequence. Though the vessels of the papillæ and deeper parts of the cutis are well seen and contain blood-cells and *débris* I have nowhere been able to find organisms in them. Nor does the cuticle, or hair substance, appear to be in the least invaded by them.

From all this it would appear that the bacilli have a strong predilection for the most superficial parts of the true skin and remain for a long time limited to this region; also that they spread superficially along the tract of the soft cells of the rete mucosum. Again, it appears not improbable that when the vesicle bursts, the production of an ordinary suppurating sore is hostile to the life of the bacillus, possibly through the introduction and antagonism of other organisms. Numerous masses of what I take to be micrococci are to be seen in the borders of the area corresponding to the eschar.

These facts, pointing, as it would appear, to the at first purely local distribution of the organisms, help to explain the now common experience of the favorable results of excision of the diseased area even many days after inoculation.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' New Series, vol. ii, p. 17.)

DESCRIPTION OF PLATE III.

(On some points regarding the Distribution of Bacillus Anthracis in the Human Skin in Malignant Pustule, by ARTHUR E. BARKER, F.R.C.S.)

Fig. 1.—Diagram, natural size, of transverse vertical section through the malignant pustule, showing central, dry, thrombosed, dark area surrounded by vesicles, and outside these the healthy skin.

Fig. 2.—Vertical section of skin through the malignant pustule. *Hartnack*, obj. 4 × 3 = × 90.

a. Horny layer of epidermis of collapsed vesicle.

b b b. Papillæ of cutis covered at their apices and sides by swarms of bacilli.

c c. Inflamed cutis infiltrated with leucocytes but showing few bacilli.

On the surface of the papillæ the rete is seen in the process of developing small vesicles, some of which have just become confluent.

Fig. 3.—Vertical section of skin. *Hartnack*, obj. 7 × 3 = × 330.

a. Horny layer of epidermis.

b. Deeper layers, with vesicles commencing to form.

c. Root-sheath of hair with bacilli descending along its boundaries.

d. A large vesicle formed by raised cuticle.

e. Clusters of bacilli located chiefly on the surfaces of the papillæ and deeper layers of the rete mucosum.

f. Clusters of bacilli in individual cells.

Fig. 3.

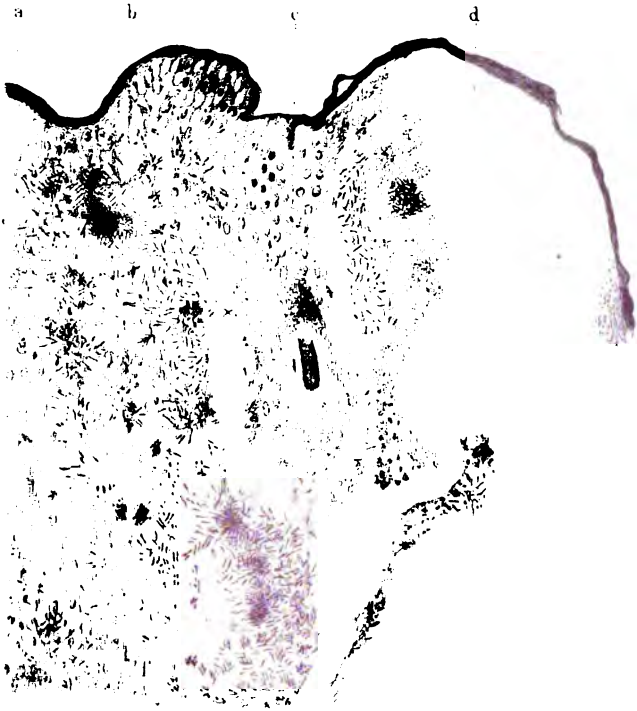

Fig. 2.

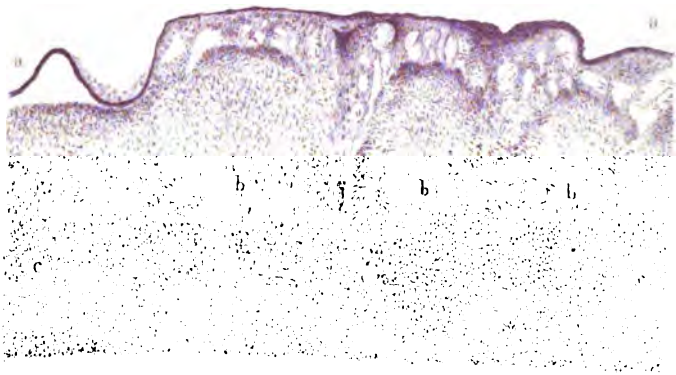

Fig. 1.

A CASE

OF SO-CALLED

ACTINOMYCOSIS OF THE LIVER.

BY

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Received November 10th—Read November 24th, 1885.

On October 1st, 1884, my friend Mr. J. Crossman, of the Wandsworth Road, London, sent Joseph Robert W— into the Arthur Ward of St. Thomas's Hospital.

The patient was thirty years old, and a joiner by occupation. He was very pallid, about five feet eight inches high, much emaciated, and weighed only seven stone. A very painful tumour, about the size of an orange, projected forwards from the left hypochondrium; the skin covering it was distended, shining, and pale; and the swelling was very painful to pressure. It was obviously connected with the left lobe of the liver, for it was limited above, below, and to the right by a hard and dull surface continuous with the liver, and it was strongly affected by the pulsations of the aorta.

The enlargement of the liver was chiefly confined to the left lobe. There was general slight impairment of chest

resonance, but the breath-sounds were fairly healthy, the only abnormality being a faint occasional crepitation at the left apex and clicking at the end of inspiration at the sides. There was neither cough nor expectoration; the heart-sounds were normal, and the impulse in the fifth space. The tongue was tender, and the epithelial covering transparent—a condition predisposing to aphtha, which, indeed, appeared very soon after and continued, with occasional recessions (from treatment), up to the time of his death. The rest of the alimentary canal remained healthy, but the digestive power was feeble.

He died ten weeks after admission into the hospital, his general condition undergoing very little change, and his weight varying only a few pounds; it attained its maximum, seven stone four pounds, about five days before his death. The temperature ranged usually, with great regularity, between 97° F. to 98° at 8 a.m., and 101° to 102° between 8 p.m. and midnight; on four occasions only the night temperature attained 103° to 103·6°.

During the last nine days of his life the temperature declined, and on the last three, instead of rising in the evening, as usual, it fell to 95°. Nocturnal (between 3 and 5 a.m.) sweating was for the first four months of his illness a troublesome symptom.

Apart from his hereditary tendencies, the patient's antecedents were good. He had had measles in childhood, but no other disease, and had led an industrious and temperate life.

The patient states that he was in perfect health seven months before his admission. A month later he came under my friend Mr. Crossman's care for an attack of acute inflammation, and he kindly furnishes me with the following information:

"*Family History.*—The father, æt. 70, has suffered for many years from asthma and chronic lung disease, and at times severe functional disease of the liver. The mother has also suffered from considerable derangement of the stomach and liver, from piles and epistaxis, one attack of the latter

being so severe as to require plugging of the anterior and posterior nares. Two sisters have been under my treatment, one dying at the age of twenty-seven years, after about six months' illness, of acute phthisis; and the other is now under occasional treatment for the same complaint, and the prognosis is extremely unfavorable. The two brothers I have not seen.

"The patient came under my care on February 23rd, 1884. He had returned from his work and was suffering acutely from 'severe pain in the bowels,' which had been preceded by shivering. There was neither vomiting nor nausea, and the temperature then, and for some days after, never exceeded 102·5° nor fell lower than 100·5°. Even when the patient lost most of the pain and fever the temperature never fell to the normal standard. During the first weeks of his illness there was an anxious expression of face; pain on moving in bed, and more or less pain over the abdomen. At one time a blister was applied over the left epigastric region (the part most complained of), and afforded relief. The base of the right lung from the first gave signs of pneumonia, and this continued for some days, and then slowly cleared up. The urine was normal in quantity and character. The liver area was normal; the heart weak but sounds healthy. In about eight or ten days his condition became chronic with intermissions and accessions of pain and feverishness. During most of the time the respirations were short and painful, accompanied with a hacking cough but with no serious expectoration. The patient always maintained a stooping posture in walking. There were no symptoms of jaundice, but a constant colourless condition of conjunctiva, much loss of flesh and great depression of spirits. After the first month there was improvement but no signs of permanent recovery, and in April, when a change was made into the country, the patient returned very little better. On May 5th, contrary to my advice, he recommenced work and continued it for several weeks. The day before he entered St. Thomas's I saw him and found for the

first time an abscess, tense and extremely painful, on the anterior surface of the liver. He was advised at once to proceed to the hospital for operation."

The swelling was characteristic of the disease. It was pallid, arose up suddenly from the parts beneath, and was surrounded by a uniformly firm base in the liver. These characters sufficiently distinguished it both from an ordinary abscess and from hydatid disease.

I incised it at once and freely, but was disappointed with the result, for not more than two ounces of pus and blood could be removed. It had a slightly offensive odour, and our house-surgeon, Mr. Makins, on introducing the finger, found that the floor of the abscess was just within the surface of the liver, which moved up and down with the diaphragm.

Drainage-tubes were inserted, and an opening maintained up to the time of his death. Great relief followed the operation, but the subsequent course showed plainly that we had to do with a lowly organised disease. The discharge was never free, and although the cavity was freely and frequently injected with aromatic antiseptics (eucalyptus and thymol) it was for a long time very offensive.

The painful edges of the wound were long in showing any disposition to granulate, and when they did so the granulations were poor and pale. Very little pus appeared upon the poultices; but a small teaspoonful of smooth, homogeneous, very thick, cream-coloured matter could at any time be extruded slowly by pressing firmly upon the indurated base of the abscess.

On the thirty-third day after admission a diffuse, painful, fluctuating tumour was discovered in the right loin. It was opened the following day, and about two ounces of offensive pus discharged; the twelfth rib, covered however by its periosteum, could be felt in the abscess cavity. Pus of the same character continued to be discharged freely for a few days, and the abscess then gradually contracted, but never completely healed.

About the time of the formations of this abscess he had a slight cough, with a little clear bronchial expectoration, and the nocturnal sweatings which had much subsided were again troublesome. On the evening of the fifty-ninth day the cough suddenly increased, and during the night he expectorated about sixteen ounces of rather offensive and slightly rusty muco-purulent matter. This was attended by signs of congestion (dulness, diminished breath-sounds, and crepitation) of the lower and hinder part of the right lung. Beyond the severe and distressing cough, there were no other symptoms. The expectoration ceased as suddenly as it appeared, and after twenty-four hours he was in his usual condition with scarcely any cough remaining.

But for the nature of the expectoration, one would have supposed that he had emptied some internal abscess by the lung. The general condition now improved a little, and once more the mouth became free of aphthæ (stomatitis fungosa—*oidium albicans* of the usual form).

The improvement, however, was only temporary. After signs of increasing weakness for a day or two the patient suddenly collapsed, and died on the seventieth day after his admission into the hospital.

Post-mortem Examination.—The body was pale and much emaciated, the abdomen not appreciably enlarged. The contour of the hypochondrium was but slightly raised, the prominency of the tumour having gradually subsided. A pale, imperfectly granulated surface, about the size of a florin, with a narrow cicatricial margin, and a central aperture admitting a No. 5 elastic catheter, were the remains of the original incision into the most prominent part of the tumour. Firm continuous pressure on the margins of the sinus caused the extrusion of a few drops of very thick creamy, homogeneous pus.

Another sinus existed in the right loin, and communicated with the old abscess cavity in that situation.

The peritoneal surface of the left lobe of the liver was

thickened and adherent to the abdominal wall in front, for an area of about two inches around the sinus, and above to the diaphragm and pericardium.

The sinus communicated with a cream-coloured, rounded, shreddy, boggy mass, the interstices of which were occupied by a thick creamy pus. The whole mass resembled a huge anthrax about the size of a large orange.

Pus could be squeezed out of any divided part, but it was for the most part retained in the shreddy interstices of the tumour.

The liver was enlarged, weighing 5 lb. 3½ oz.; its substance generally was quite normal. It stained black when soaked in 1 per cent. solution of osmic acid; the bile and fæcal matters were typically healthy in appearance. A number of globular masses of morbid deposit were scattered through the gland, two of them being nearly as large as the one which had pointed externally; several were of the size of Tangerine oranges; the smallest were aggregations of a few tubercles the size of hemp seeds. The smallest and youngest were co-extensive with the hepatic lobule, and they were almost as soft as brain substance. Where a dozen or more such tubercles were aggregated the intervening liver tissue was replaced by a coarse soft stroma, white and shreddy, but near the surface often discoloured by post-mortem staining. Sections of these smaller tumours presented an appearance exactly similar to that of caseous tubercle in red hepatised lung.

The larger masses were always spherical, and their central portions more or less softened; being somewhat confined by the surrounding liver, they bulged a little beyond it when they lay near the surface.

These tubercular masses were scattered throughout the liver, the larger and more advanced being in the thickest part of the gland, and here two of them, each nearly three inches in diameter, were separated by a band of liver barely a quarter of an inch thick.

The youngest of the morbid deposits were found in the thinner and marginal parts of the gland.

The disease was thus seen in all its stages from the invasion of a single lobule of the liver, to the large purulent mass which had been incised.

The liver-substance immediately surrounding both large and small masses was dark and congested, and this exaggerated what would have been otherwise a very sharp line of demarcation between the healthy and morbid structures.

The diaphragm was adherent to the surface of the liver by recent inflammatory action. A few scattered yellow tubercles the size of hemp-seeds pervaded both lungs.

The right lung weighed 1 lb. 14½ oz., and by its base was adherent to the pericardium.

The left lung weighed 1 lb. 7½ oz. Both lungs were œdematous.

The pericardium was the seat of a chronic inflammation; it was thickened and adherent both to the pleuræ and diaphragm—to the latter in the immediate neighbourhood of the incised mass; and here it was reddish as if sharing in a continuous inflammation. The cavity contained 25 ounces of serum, and both visceral and parietal layers were thickly covered with a shaggy lymph. The heart weighed 13½ oz. and was quite healthy.

With the exception of the vermiform appendix, the intestines were healthy. The appendix was long and wide, and lay turned up along the attached part of the ascending colon. Here it was inflamed and adherent to the abdominal wall, which itself formed the limits of the lumbar abscess. I am doubtful whether there was any communication between them, there was certainly no trace of pus in the appendix, the summit of which contained a little soft fæcal matter.

The kidneys were rather large, weighing together 15 oz., but they were apparently normal in structure, as was the spleen (9½ oz.) and the rest of the organs.

Minute Examination of the Liver.—Sections preserved in spirit are extremely instructive and interesting. The morbid masses are distinguished by their paler, almost white colour, and a netted appearance (Pl. 4, fig. 1). In the smaller and younger masses the apertures of the network—*cavities*, as I will call them, are circular, average the one twenty-fifth of an inch in diameter, and are regularly placed, the intervals being usually equal to the width of the cavities. In sections of the older masses many of the cavities are larger, some the eighth of an inch broad, and are evidently formed by absorption of the partitions. Some of the cavities are elongated and more or less acutely elliptical or slit-like, sections, in fact, of bending tubes.

Many of the cavities appear as mere cup-shaped depressions, others are deep and winding; all but the smallest present secondary depressions or rounded ridges, sometimes faintly, sometimes strongly, marked; they also present a number of minute pin-hole apertures upon their walls, but sometimes the cavities communicate by wide openings. The stroma or framework of the morbid mass is composed of the thick walls of these cavities and their intercommunicating passages. It is a compact, dense, fibro-elastic tissue, yellowish white where it lines the cavities, but greyish and faintly diaphanous in the intermediate portion. This stroma forms everywhere a complete investment, being continued around the mass as a sinuous border, soon blending with the liver substance and streaking it as it does so with faintly marked concentric lines.

It is clear from this description that the framework of the morbid mass contains within its walls a system of rounded cavities freely communicating throughout by fine, and occasionally by large, passages; in brief, it is a close network of fine thick-walled tubes, presenting comparatively wide dilatations or cavities at frequent and pretty regular intervals;—a structure approaching that of ordinary erectile tissue.

The question at once arises, what is the origin and

what the relationship of this network of enormously thickened vessels?

Sections taken from any part of the liver show the hepatic canals (Pl. 4, fig. 1, *b*), and also the sublobular veins to be perfectly healthy, even when the former lie within half an inch of the main foci of the disease, and the latter ramify within its area. But the reverse is the case with the portal canals; both arteries and veins are everywhere enormously thickened, and the intervening connective tissue proportionately increased (Pl. 4, fig. 1, *c*). Further, these thickened vessels could be traced into direct continuity with the network of vessels which forms the stroma of the tubercular mass. It thus appears that the afferent vessels—the portal vein, and the hepatic artery, are those which are engaged in the morbid process; the hepatic vein escaping any implication.

Whatever share the lymphatics may have had originally in the morbid process, they appear to have no place in the dense, almost tendinous tissue in which the vessels are now embedded. The bile-ducts also appear to be obliterated. Of the two vessels, the portal vein and the hepatic artery, thus associated with the disease, it will doubtless be conceded that it is the artery which takes the principle share in the process. Yet it is not certain that any new vessels are formed; I do not think it is necessary to assume so, for the main bulk of the vascular stroma may be regarded as the confluent interlobular plexuses of the morbid areas. The cavities, however, have a different origin, these I regard as the thickened capsules of the invaded hepatic lobules—each of the smaller cavities representing an excavated lobule, its wall being formed of the hypertrophied connective tissue of the interlobular spaces, and perforated by the branches of the interlobular plexus, which naturally enter the lobule. Thus is formed a network of blood-vessels of an average diameter of the $\frac{1}{100}$th of an inch, communicating freely with little cavities continuous with them, measuring about the $\frac{1}{33}$rd of an inch in diameter. As the disease advances to its purulent

stage these cavities may be enlarged by dissolution of the intervening walls.

Further proof of this view of the origin of these cavities is furnished by microscopical examination (see p. 145).

I proceed now to describe the contents of these cavities—these sites of the original hepatic lobules. Turning again to the sections preserved in spirit, and using a slight magnifier, it will be observed that these little spaces are partially filled (Pl. IV, fig. 2), each by a little yellow, glistening, rounded *granule* lying naked in the recess, or partially embedded in a little soft matter which is easily washed away by a drop or two of water. The larger cavities, those formed by confluence are usually occupied by aggregations of these granules, which often resemble in contour a microscopical raspberry.

These minute granules vary much in size, the smallest are scarcely visible to the naked eye, while the largest sometimes attain the $\frac{1}{10}$th of an inch in diameter; the majority are about the $\frac{1}{40}$th of an inch (Pl. IV, fig. 3).

Characters and Structure of the Granules.—As may be inferred from the above description, the granules lie loose in the cavities containing them, and they may be readily shaken or picked out of the cells ("cavities") which are exposed in the section. Availing myself of this fact, I have been able to collect and examine them thoroughly. They are of a straw-yellow colour to the naked eye, but under the microscope they are often stained of a deep brown colour; they are spherical, oval, pyriform, reniform, and even subangular in outline, and obviously composed of aggregations of smaller granules about $\frac{1}{600}$th of an inch in size. Each constituent granule has a smooth continuously curved surface, but the aggregation is convoluted like a nodule of hæmatite, and like many renal calculi they present sometimes one or two nipple-like elevations. Exposed to the air they turn of a rich brown colour on drying, they are quite solid and apparently quite homogeneous, and have an average sp. gr. of 1·25; they have the consistence

of soft cheese, being friable, and easily compressed by the microscopic covering glass; many, however, give indications of slight grittiness. They stain well and easily, both with watery and alcoholic solutions of the dyes, and they become dark in 1 per cent. solution of osmic acid. Treated successively with nitric acid and ammonia they give the xantho-proteid reaction. Thus treated and disintegrated a number of oil spherules are set free. Exposed to combustion, they shrink very much, and leave a small quantity of white ash, soluble in dilute HCl and giving when neutralised a precipitate with oxalate of ammonia.

It appears, therefore, that they are composed of a proteid substance associated with a little fat and calcic carbonate.

Microscopical Structure of the Morbid Deposit.—Sections of the morbid area showed that here the hepatic lobules were in some places completely occupied by leucocytes, and in others by leucocytes with the granules above described (Pl. VI, fig. 2). The interlobular spaces were sometimes obliterated by the coalescence of the lobules, and sometimes they formed very wide bands of nucleated connective tissue pervaded by dilated, and often varicose, thick-walled vessels, sometimes loaded with red corpuscles. Thus wide barren fields, the $\frac{1}{25}$th of an inch and sometimes more, composed wholly of leucocytes to the complete outcrowding of liver-cells and blood-vessels, were presented to the view (Pl. VI, fig. 2). The leucocytes were well formed—granular spherical corpuscles varying from the $\frac{1}{3500}$th to the $\frac{1}{2500}$th of an inch in diameter, the majority being the $\frac{1}{3000}$th. In the older tubercles these corpuscles occasionally presented degenerative changes, becoming clear and glistening, and staining imperfectly (Pl. VI, fig. 1, *a*).

The appearances described were in successful sections prettily varied by the *granules* (see Pl. V), which formed bold groups of islands in the general waste of leucocytes, for they are composed of a denser material, and present

in section a radiated structure like concrete crystals of calcic carbonate (Pl. V, VI).

The usually aggregate condition of these bodies is well seen in sections. The simple spherical granules of which the majority are composed vary in size from the $\frac{1}{800}$th to the $\frac{1}{300}$th of an inch, but in the progress of the disease do not long remain isolated. In section the larger composite granules have sometimes an angular outline flanked by rounded bastions (Pl. V, fig. 1).

The granules are embedded in and adherent to the surrounding leucocytes, but there does not appear to be any continuity of structure between them, for the granules readily fall out of the sections, and after rinsing in fluid present a very smooth surface. Still in fresh specimens the adhesion is tolerably firm. In the older tubercles, where the leucocytes have begun to soften, it is difficult to retain the granules in sections, and their place is usually occupied by a wide lumen.

Under a low power (× 120) sections of these simple or composite granules present a radiated structure, in some faintly indicated, in others very distinct. The centres of some are diaphanous, or even luminous, the lumen being circular (Pl. VI, fig. 1), or from pressure subangular. Some of these openings are the $\frac{1}{800}$th to the $\frac{1}{1000}$th of an inch. The centres of other granules are dense and prevent the passage of light. Usually, however, the centres are lighter than the rest of the granule, and present an irregularly netted appearance (Pl. VI, fig. 1), as if due to a fine scanty stroma, which stains more readily than the adjacent tissue. The radiations proceed from the central clear space, or the apparent nucleus, with regularity, as straight or occasionally very slightly curved lines, and terminate without alteration in the surface of the granule, and thus impinge upon the leucocytes which are adherent to it. Under high powers, and when every detail in the structure of the leucocyte is clearly defined, the radiated masses gain nothing in appearances. The radiations remain soft, glistening, and wanting in sharp outline.

The netted centre which I have described above as stroma is in some granules more clearly seen than in others (Pl. VI, fig. 1).

Twelve or more of these granules, some simple, some composite, are frequently seen forming patches or colonies occupying a considerable portion of the site of a lobule (Pl. VI, fig. 2). For a time they are separated by the intervening leucocytes; as, however, they enlarge and coalesce, the leucocytes undergo degeneration; they wither, and, if they do not pass into pus, become reduced to a diaphanous tissue, sprinkled with fine molecules, and difficult to stain (Pl. V, fig. 1).

Changes also occur in the granules themselves. As they grow older and larger they present a thick clear cortical portion, destitute of striation, which, commencing apparently upon its surface, may be occasionally seen stretching far away into the tissue formed by the degenerating leucocytes (Pl. V, fig. 2). The morbid deposit in the lobules of the lungs presented exactly the same features, but here the action was more limited, being confined to single lobules.

Pathology.—It would appear that the first step in the morbid process is the extrusion of leucocytes. Is it a mere arrest of them in the liver, or is the lymph tissue in this organ too active in generating them? Of these two suppositions, the former is perhaps nearer the truth, for we know that the liver, like the lungs, is constantly receiving large numbers of leucocytes, and as they do not pass out of the efferent vessels of these glands we must assume either that they are used up in the chemical processes going on in these glands, or that they are converted into red corpuscles. If the latter be the case, then it is easy to explain the plethora of leucocytes in the hepatic capillaries, by assuming a diminution of the oxydising processes—a diminution of arterial blood. The question suggest itself: Would partials ligature of the hepatic artery result in the development of tubercle in that gland?

Whatever may be the cause, a plethora of leucocytes

is one prominent fact, and, apart from any obstruction to the hepatic artery, we can understand how a plethora of these white corpuscles, by outcrowding the red, and standing between them and the liver-cells, would lead to a depression of the chemical action in the liver.

As an effect of the foregoing plethora and subsequent effusion of the leucocytes, the liver-cells wither and ultimately disappear, together with the intralobular plexus of blood-vessels. Severe congestion of the interlobular plexus is the result in these areas at first; then follows, with increasing obstruction, dilatation and thickening of these vessels; and when the obstruction in the lobules is complete, stasis and, perhaps under the attendant irritation, plugging. In a large branch of the portal vein I detected an old clot sending branches far and wide into the small lateral vessels. Under the microscope this shrivelled clot was seen to be spangled with colourless crystals of calcic carbonate in spherical radiated masses, and in aggregated prisms.

In marginal sections of the diseased liver the smallest arteries are seen to be early affected. Leucocytes invade their walls and stand in single and double file around them; while others are stationed between the rows of liver-cells.

If the view which I have taken of the formation of the cavities of the stroma be the true one, it follows that the granules are formed in the interior of the lobules. When the leucocytic invasion of these is complete the blood current is of course entirely cut off, and the central parts of the lobule, being farthest removed from nutrition, show the first indications of degenerative change.

The deposit of a little calcic carbonate in the nucleus of a leucocyte may be the starting-point of the granule, its subsequent development being due to the extension of the calcareous deposit into the surrounding tissue, the leucocytic surrounding furnishing nutrition to the growing granule just as the mucous membrane supports the growth of a urinary or biliary calculus.

Whatever the morbid action may be, there can be no doubt, I think, that it originates in the lobule, for it is here that its effects are most obvious, while they are at the same time farthest removed from the first stages.

When these tubercular masses soften down, the pus is of course wholly contained in the vessels of the stroma. In the early stages the vessels, for the most part at least, remain pervious and partially filled with leucocytes, escaped, we may assume, from the lobules.

In the later stages they are filled with pus, and the difficulty of evacuating this is explained by the fact that in every cavity there is a granule, and sometimes in the apertures of that cavity a corresponding number of nipple-like projections from the granule: the smallest and simplest of these granules forming therefore a great, and the larger and more complex ones a complete, obstruction to the outward flow of pus.

Having now finished my history of the case, I pass to the consideration of a question of great interest in reference to the disease which I have described.

Those who are acquainted with the history of *actinomycosis*, and have heard my story and looked at my illustrations, will be ready to say, "It is a genuine and typical case of actinomycosis."

I am bound to admit that it agrees in many particulars with most of the typical cases of this disease which have been recorded, and my figures correspond exactly with those of Lebert,[1] Israel,[2] and others, and yet I am perfectly satisfied, and hope to prove to the Society, that there is no fungus whatever necessarily associated with my case. If this be so, then much if not all of the so-called actinomycosis disease must be relegated to its old, and, as I believe, its proper place, namely, "tubercle."

There can be no doubt then that we have under consideration an example of what has been described and illustrated by several authors as actinomycosis, and it is

[1] Traité d'anatomie pathologique,' Atlas; Tome i, pl. ii, fig. 16. Paris, 1857

[2] 'Archiv für path. Anat. und Physiol.,' Virchow, Bd. 74, 1878, Taf. ii, iii, iv.

necessary that I should state the facts which lead me to reject the fungus theory of the production of the disease.

It will be conceded that the present case furnishes a complete illustration of the disease from its first origin as a few escaped leucocytes in the centre of a lobule of the liver, to the ripe, purulent mass which projected externally. If the disease be due to a fungus, the fungus is here accessible to our observation and readily capable of demonstration. Simpler still, the fungus is confined to the granules, and it is these,[1] therefore, to which I must invite attention.

These granules may be regarded as typical examples of caseous degeneration of tubercular deposit.

I have stated that they are composed of a solid albuminous matter containing a little fat and calcic carbonate. The inorganic matter has been very long recognised as a constituent of tubercular nodules, and when it is in sufficient abundance to make them gritty, there is no denying its presence. But I am not aware that the advocates of the fungus origin of this disease will allow that any portion of the radiation in such a case as I have described is due to crystalline structure. They regard the rayed appearance as being due to the club-shaped asci of the fungus. In the present case nothing is easier than to disprove this view. If a section of a granule, or an aggregation of them, be selected for the boldness and distinction of its rayed appearance, and treated with strong acetic acid, while it is observed under the microscope, the radiations will melt away rapidly and, except perhaps in an old granule here and there, completely disappear, thus proving that they are due to crystalline matter soluble in the acid. It is in fact a delicate impregnation of an albuminous and fatty basis with calcic carbonate, which, like the organic basis of bone, may be removed without affecting the integrity of the matrix in which it is deposited.

[1] A large number of these isolated granules were exhibited to the Society.

This simple test is decisive, for if any fungus were present its finest portions would be brought out conspicuously in a specimen cleared by strong acetic acid.

Granules or their sections may be rendered perfectly transparent and subsequently disintegrated by means of acetic or the mineral acids, by caustic potash and ammonia, and when examined in this state by the highest powers ($\times \frac{1}{1800}$) they have failed to furnish me with the faintest trace of fungoid growth.[1]

Turning now to the physical conditions of the fungus, let us see what presumption these afford of the presence of a fungus. First, as to its position in the body. We find it in a flourishing condition, according to the descriptions, in the very centre of the morbid mass, where it is bathed in carbonic acid, and shut off from oxygen—a condition, as far as we know, incapable of supporting the growth of a fungus, which more than all other vegetables wants a free access of oxygen.

Again, the granule is not a mere mouldy mass like a bit of mouldy cheese, with its cavities, cracks, upheavals, and erosions, but a compact solid body with a smooth surface like a nodule of hæmatite. Cut it which ever way we will, we fail to recognise sections of the filaments or asci, which, if any such existed, would be, according to the measurements given of them, as plainly visible as the cross sections of fibres in a medullated nerve-bundle. The outer ends of asci are represented as not being all on the same level at the circumference of the actinomycosis mass, but my granules give no indication of such irregularity; they have, as I have said, a smooth and rounded surface.

Having examined the youngest and oldest of the isolated granules with the same result, I have explored a large quantity of debris, obtained by washing out the cells of the stroma with spirit.

This debris was composed (*a*) of granules; (*b*) of whiter

[1] See Appendix.

and lighter flocculent masses of leucocytes, in which the granules were embedded, and (*c*) a very small heavier residue composed of crystals. No trace of fungus was found in the lighter portions of the debris. The crystals were very minute, none more than the $\frac{1}{500}$th of an inch in size, and as they all dissolved in acetic acid with escape of bubbles of gas, I assume that they were all calcic carbonate; a few were thick and rhomboidal like Iceland spar, a few others were smooth, spherical, or elliptical masses, the majority were clusters of a few coarse or many fine prisms. Some of the latter were beautiful rosettes, and when treated with acetic acid they separated into their constituent prisms, which had a strong resemblance, on account of their clavate form, to the conidia or asci of the actinomyces.[1] Sometimes two crystals were united, causing a forked appearance, which gave a still stronger resemblance. Soon, however, they melted in the acetic acid and totally disappeared. All these crystals were bright and colourless.

Scanning the field, on one occasion, with a very high power and a too thick covering glass, I caused it to slide as I was passing over some thin plates of cholesterin, when all at once the looked-for fungus, as I thought, appeared. Everywhere in the field long distinct filaments with expanded ends lay in bundles, and on all sides arborescent and feathery forms.

I mention this because, if a similar displacement had occurred in a fragment of cholesterin overlying one of the radiated masses, its meaning could only have been interpreted by the use of a solvent, of which there are so few for cholesterin.

Are we now to assume from this case that fungi are secondary and therefore non-essential developments in the cases of actinomycosis which are recorded. This, I think, would not be assuming too much. Fungi may spring up anywhere in the body when there is a free surface and a supply of oxygen, or in any fluid of the body, and there

[1] Israel, 'Virchow's Archiv,' 1878, t. iii, fig. 5.

is perhaps no more likely place than the sinus of an old abscess—nay, more, the surface of the granules themselves when they are thrown out into the sinuses may become clothed with fungi. A patient of mine died of phthisis many years ago in Kings' College Hospital, and at the post-mortem examination two of the papillæ of one kidney were found ulcerated; on examination I found the *Oidium albicans* luxuriantly developed for some distance along the straight tubules. But the conditions in such cases, as I have just mentioned, are very different from those of the so-called actinomycosis, in which the fungus is assumed to develop in a solid mass without disturbing it.

The striations which I have described and figured are, I maintain, nothing more than the earliest indications of that calcareous and fatty degeneration to which caseous tubercular deposits are so liable, and have no more connection with fungoid growth than a gall-stone has.

Appendix.

On the occasion of the reading of this paper my late Demonstrator of Physiology, Dr. Theodore Acland, who has taken a most laudable interest in this case, exhibited some specimens of mycelium obtained from it which he observed only two or three days previously when he was looking for bacilli. After the lapse of a year from the death of the patient, I naturally concluded that the fungus was a post-mortem development. Nevertheless, I have thought it my duty to reinvestigate the matter. Knowing how prone such matters as caseous tubercle are to fungous invasion, I was careful in making my original investigations to select the smallest and youngest of the tubercular masses, and to avoid those which had any communication with the external sinus, which had existed for many weeks and was frequently injected with fluids from without. In these, as I have stated, I have failed to detect any trace of a fungus.

In renewing my search the only material left to me was the museum specimen and the slice which is represented in Pl. IV. This includes the ripest portion of the disease and that which lay in contact and continuity with the incised mass, and also some of the youngest deposits as seen at *d*, Pl. IV, fig. 1. The specimen had been kept immersed in methylated spirit in a glass dish, covered loosely by a plate of glass, and it had been drained and exposed upon a glass plate several times for the purpose of examination and delineation. It is this portion of the liver which I have examined. I took the granules promiscuously, removing some from their natural position in the cells of the stroma, and collecting others which had fallen out into the preservative fluid. They were stained and mounted by the most approved methods for demonstrating micro-organisms.

In this way I have examined great numbers of these granules, and the result is that in a very few I have found traces of an extremely fine mycelium-like structure, but none of the club-shaped asci which are regarded as characteristic of the *Actinomyces bovis*.

Now, under the circumstances it will be conceded, I think, that the complete absence of fungoid growth would have been more remarkable than its presence, and this renewed examination has confirmed me in my former opinion that the fungus is not of the essence of the disease, but merely an occasional and accidental associate. With due deference to those who regard the fungus as the essence of the disease, I would ask them, as opportunities occur, to direct their attention to those portions of the diseased structures which have no communication with the surfaces of the body, and to the very earliest developments of the morbid action, and by this means exclude the question of accidental and secondary contamination.

The case above narrated is, I believe, the first of the kind which has been noticed in this country, and it is certainly not a common form of disease. I have regarded it from the first as an example of tubercular disease from

which the liver is so remarkably free; and the close examination which I have given the case confirms me in this view. (*May*, 1886.)

(For a report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' New Series, vol. ii, p. 20.

DESCRIPTION OF PLATES IV, V, AND VI.

(A Case of so-called Actinomycosis of the Liver. By JOHN HARLEY, M.D.)

PLATE IV.

Fig. 1.—Section of the liver as it appeared in methylated spirit (natural size).

(*a*) One of the principal masses.

(*b*) Hepatic veins.

(*c*) Portal canals; vessels much thickened.

(*d*) Youngest deposits.

Fig. 2.—A portion of (*a*) Fig. 1, showing the cavities, some containing granules. × 3.

Fig. 3.—A heap of isolated granules. × 2.

PLATE V.

Figs. 1 and 2.—Radiate granules, surrounded by leucocytes. × 60.

PLATE VI.

Fig. 1.—A minute composite, radiate granule, showing variations in the central parts; in one a circular lumen, in others a nuclear matter, and in the largest a netted stroma. This granule is surrounded by leucocytes, some of which (*b*) are partially, and the rest (*a*) wholly, degenerated. × 150.

Fig. 2.—Three lobules invaded by leucocytes (*a*, *a*), interspersed with radiate granules, darker, and separated by thick walls of fibrous tissue, containing thick-walled blood-vessels. × 12.

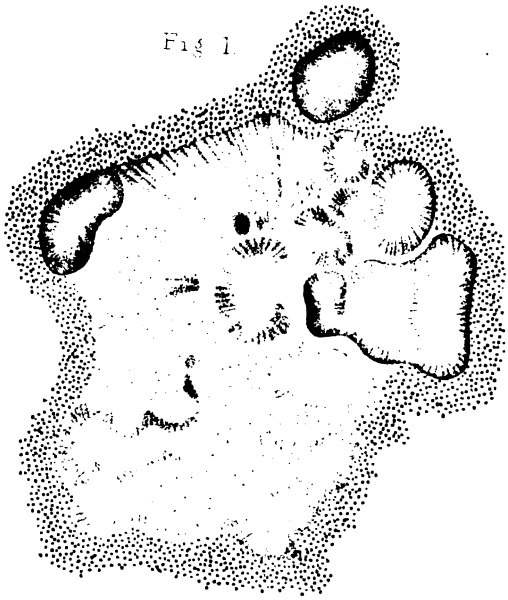
Fig. 1.

Fig. 3. × 2.

Fig. 2. × 3.

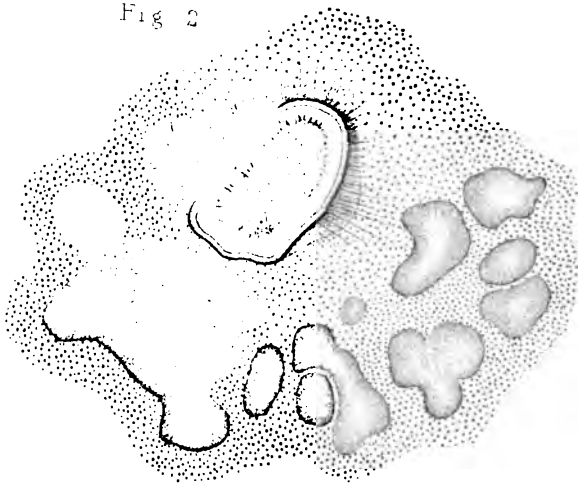
S.M.Harley ad nat del. Mintern Bros Chromo lith.

Fig 1.



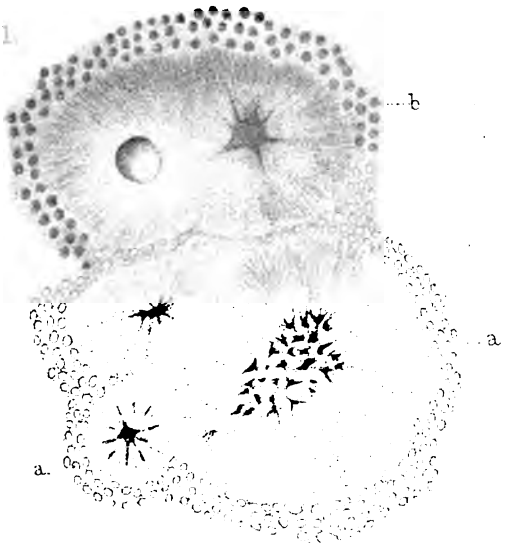
x 60

Fig 2.



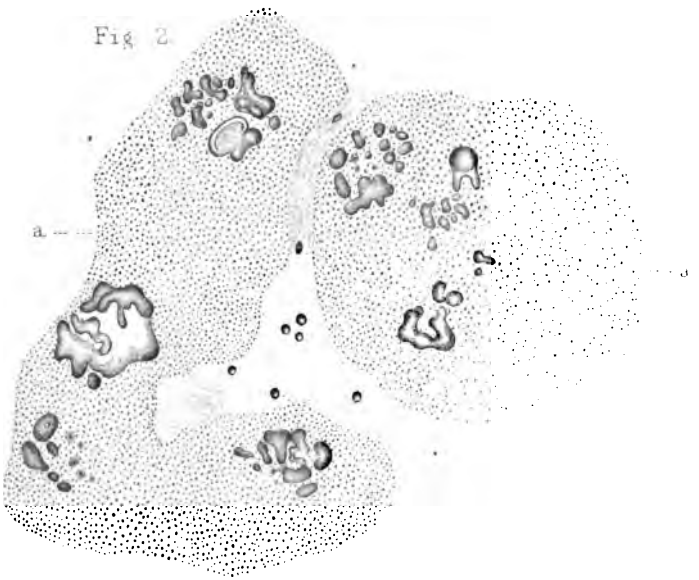
x 60

Fig. 1.



x 150

Fig. 2.



x 12

A CASE

OF

DESTRUCTION OF A PORTION OF THE AXILLARY ARTERY

BY

SARCOMA.

BY

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Received May 15th—Read December 8th, 1885.

A LABOURER, aged thirty-three, a fine, powerful man, came to the hospital in November, 1884, with a large tumour in front of the chest on the right side, plainly visible by its prominence, although situated beneath the pectoral muscles. It extended from beneath the clavicle to the axilla, where it could be seen and felt with a well-defined border, immediately behind and somewhat beyond the lower margin of the pectoralis major. The mass was uniformly soft; to some suggesting even fluid, but to most of us a texture like fat or cellular tissue. There was no pain or any material uneasiness in the part, but the pulse in the arm of that side was much smaller than in the opposite one. The man himself had been aware of something wrong for about nine or ten weeks, and during the fortnight or so that it was under observation the tumour manifestly increased.

It was decided to attempt the removal of the growth. I exposed its outer extremity by a free incision along the lower margin of the pectoralis major where it appeared, through the fat of the axilla, by the well-defined surface of a distinct capsule, but a little further dissection clearly showed it to be a soft sarcoma. The pectoralis major first, and then the minor, were divided so as to reach the upper portion of the tumour, which extended to the large vessels, and was found completely investing them for at least some three or four inches of their course. All that part of the tumour which lay below the vessels was easily removed, but it was determined to make no attempt to detach the portion which invested the vessels, and we proceeded to secure some small and insignificant arteries which had been divided in the operation. While thus engaged it was observed that the hæmorrhage, which up to that time had been but slight, began to increase considerably from the region of the upper portion of the tumour, but no particular vessel, as its source, could be distinctly seen. However, even every touch with the sponge seemed to make matters worse, and in a few seconds more there was such a gush of arterial blood that it was with the utmost difficulty controlled by Mr. Marsh, who dexterously grasped the bleeding mass. An endeavour was made to assist him by pressure on the subclavian above, but this had very little or no effect. In order to obtain a clearer view of the bleeding orifice I exposed for a short distance the axillary vein, a small part of which could be just seen, placed two ligatures on it, divided it between them, and turned the ends up and down. Then we could discover no artery in the situation where the axillary ought to have been found, but it was plain that the blood came from the place which it should have occupied—both from above downward and from below upward. After two or three ineffectual attempts, I succeeded in grasping the upper orifice with pressure forceps, which arrested the hæmorrhage in that direction, but the abundant hæmorrhage from below still continued

until the lower orifice was in like manner secured. When the immediate danger from this cause was over we could with more leisure secure two or three other bleeding points in the immediate neighbourhood by additional forceps, but no ligature would hold, and after one or two futile attempts to apply them, we were compelled to leave the forceps as they had been placed on the vessels. Around them, for additional security, some strips of lint, soaked in a solution of perchloride of iron, were carefully packed, and the wound was partially closed. It had become evident to us all that the integrity of the main artery had been destroyed by the disease; for in no other way could the furious hæmorrhage be explained, as the knife had never been used in that region at all, and when the vein was divided no trace of the vessel in its place could be found.

The man was in a state of collapse for some time, but he gradually rallied, and for just a week after the operation he went on as well as possible. There was no sign of any recurrence of the hæmorrhage, and his only complaint was of some numbness in the tips of one or two of the fingers. But then, on a sudden, there was a violent gush of blood from the wound, and before it could be arrested the man was dead.

The axillary artery was traced from below upward in a natural state, until it arrived at the substance of the tumour, into which it passed. When this was laid open, an irregular aperture was found in the artery just above the lower border of the tumour, and from this point upwards, for another few lines, the artery was completely broken up and rapidly disappeared, so that, for about two and a half or three inches, no further trace of arterial wall could be discovered. The boundary of the cavity beyond, through which the blood must have passed, appeared to be simply the substance of the tumour, until at its upper part, just below the clavicle, arterial wall was again found, and this was continued, surrounded by the tumour, into the subclavian artery.

The substance of the arterial wall, especially of its lower portion, was infiltrated with the sarcomatous growth, and was thus rendered soft and easily lacerable. Round cells, in abundance, were crowded through the whole thickness of the arterial tunics. The termination of the artery, below and above, in the tumour was very indefinite. The tissue of one blended with that of the other, so that it became impossible to define exactly where the artery ended and the growth began. The lower portion of the artery, for an inch and a half from the orifice, was occupied by firm pale clot, evidently of some duration.

Mr. D'Arcy Power has been good enough to favour me with the following note of the histological appearances presented by the axillary artery at a point immediately below the seat of rupture.

"The artery is embedded in a mixed-celled sarcoma, which has infiltrated the tunica externa in such a manner as to render it impossible to separate the vessel from the tumour. The middle coat is thickened by an increase of its fibrous tissue, and intermixed with the elastic fibres are a large number of sarcoma cells, most of them round, others fusiform. A distinct band of sarcoma tissue occupies the centre of the middle coat. The internal coat is reduced to a thin elastic membrane, which has, in some places, given way, thus allowing the sarcomatous tissue to extend into the lumen of the vessel. The same changes are visible in sections of the thoracic axis."

The axillary vein, which had been divided in the operation, was found to be but little altered—perhaps somewhat dilated where it passed through the substance of the tumour; but there was no breach of its continuity. It was filled with recent clot.

The nerves of the brachial plexus were found in a normal state.

The tumour itself presented all the characters of a round-celled sarcoma.

Another case like this is not within my experience, nor can I find a similar one on record. Of course, in-

stances of malignant tumours—both sarcoma and cancer—and others, involving large vessels, and even completely including them, have been frequently met with. Nay, instances are not very rare in which such vessels, by such means, have been seriously obstructed, and even penetrated or otherwise much damaged by the invasion of the growth. But here a considerable portion of the axillary artery was completely destroyed, and, for more than two inches, the blood stream must have passed through a channel whose walls were formed of the substance of sarcoma only. I suppose it would be generally affirmed that the arterial tunics are remarkable among tissues for the resistance they offer to destructive action of any kind. We all are familiar with cases in which they have been seen traversing long tracks of disease that has destroyed the surrounding structure, still in their integrity. There are indeed, I need not say, notable exceptions to this. For one, I may refer to a case recorded in the sixty-fourth volume of our 'Transactions,' in which several inches of the common carotid artery, as well as of the jugular vein and pneumogastric nerve, had disappeared in an abscess. But the present case is remarkable, and to me singular, in that there was not only complete destruction of a large portion of an artery, and this by a malignant growth, but that no other structure invaded by the tumour appeared to have suffered in any considerable degree.

The specimen is preserved in the museum of St. Bartholomew's Hospital.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' New Series, vol. ii, p. 25.)

AMPUTATION AT THE KNEE-JOINT BY DISARTICULATION;

WITH REMARKS ON

AMPUTATION OF THE LEG BY LATERAL FLAPS.

BY

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Received August 31st—Read December 8th, 1885.

AMPUTATION by disarticulation at the knee-joint was first performed in England by Mr. S. Lane at St. Mary's Hospital in 1857 ('Lancet,' 1857, vol. ii). The operation was first prominently brought before British surgeons in an able paper by Mr. G. D. Pollock[1] and more recently by Mr. P. Pick, in an interesting communication read before the Medical Society of London.[2]

I have practised the operation since the year 1868. In America it has found able advocates in Dr. Stephen Smith, of New York,[3] Dr. Markoe, of New York,[4] Dr. John H. Brinton, of Philadelphia,[5] and Dr Staples.[6]

[1] 'Med.-Chir. Trans.,' vol. liii, 1870.

[2] 'Med. Soc. Proceedings,' vol. vii, 1884.

[3] 'New York Journal of Medicine,' Sept., 1852, and 'American Journal of Medical Sciences,' January, 1870.

[4] 'New York Medical Journal,' January, 1856, and March, 1868.

[5] 'American Journal of Medical Sciences,' April, 1868.
Ib., January, 1872.

Yet, in spite of this advocacy, the operation is not frequently performed. By the majority of surgeons it is still regarded with suspicion.

It is difficult to estimate how far this dislike of the operation is due to a want of experience of its advantages and how far to the groundless dread of leaving articular cartilage upon the bone, under the mistaken impression that it will probably undergo degenerative changes, and so retard repair. I would also give, as an additional reason for the neglect of the operation, the personal liking which surgeons have recently shown for what I, for the sake of clearness, prefer to call the condyloid operation of Velpeau, or the supracondyloid amputation of Stokes.

It is clear that the operations of Velpeau and Stokes, are applicable to cases of disease or destruction of the knee-joint itself, whereas the operation of amputation by disarticulation at the knee-joint can only be performed when the disease, or injury, for which the amputation is practised is localised to the leg; when the condyles of the femur are unaffected or but slightly involved; and when there is a sufficiency of healthy soft parts below the knee, from which good flaps can be made. With these conditions present, the operation of amputation by disarticulation should, for reasons to be given presently, be performed.

I will now proceed to consider the value of the operation as shown from my own practice.

Table of Cases of Amputation at the Knee-joint by Disarticulation.
(Pathological Amputations.)

Date of operation.	No. of case.	Name of patient.	Age.	Disease or injury.	Nature of operation.	Result.	Remarks.
Sept. 15, 1868	1	Edward L—	44	Extensive necrosis of the tibia	A single long anterior flap; patella removed	Good	Excellent stump. Soft parts moveable over condyles. No sloughing of flap.
April 13, 1869	2	Mary A. B—	8	Sarcoma of the tibia	A single long anterior flap; patella removed	Good	Anterior flap sloughed, and exposed one condyle of femur, from which the cartilage exfoliated. The child died in three months from a return of the growth in the stump.
Jan. 16, 1870	3	Thomas S—	48	Osteo-arthritis of knee. Limb like a flail, and disorganised	A medium-length anterior flap, with a long posterior flap; patella left	Good	An excellent stump was obtained. No sloughing.
Jan. 14, 1870	4	Thomas N—	16	A useless stump after an amputation of the leg for injury	Long anterior flap and long posterior flap; patella left	Good	Immediate union of the wound. Good stump.
Aug. 9, 1871	5	Eliza L—	38	Elephantiasis of foot, with extensive ulceration exposing bone	Long anterior and posterior flaps; patella left	Good	Excellent stump. No sloughing. Cicatrix in hollow of condyles and well behind bone. In 1884 stump perfect.

Date of operation.	No. of case.	Name of patient.	Age.	Disease or injury.	Nature of operation.	Result.	Remarks.
Dec. 4, 1871	6	James S—	65	Cancer of stump of leg, which started in cicatrix of amputation performed years before	Long anterior and posterior flaps; patella left	Good	Excellent stump. Integument moved freely over condyles.
May 7, 1872	7	Samuel H—	50	Cancer of leg, with necrosis of tibia	Long anterior and posterior flaps; patella left	Good	There was slight sloughing of the anterior flap, but a good stump, which a year later was pronounced "excellent."
April 19, 1872	8	John T—	32	Epithelial cancer of leg and tibia, with necrosis	Long anterior and posterior flaps; patella removed	Died on 4th day	Died on 4th day from shock. Diseased kidneys.
July 28, 1874	9	Daniel B—	21	Disease of knee of 15 years' standing, with displacement backwards	Lateral flaps; patella left	Good	Rapid recovery. Excellent stump.
Mar. 29, 1874	10	Minnie H—	13	Old disease of knee	Lateral flaps	Good	No sloughing.
Nov. 7, 1873	11	Caroline A—	26	Acute necrosis of tibia, with disease of ankle- and knee-joints	Lateral flaps; patella left; surface of one condyle taken away where the cartilage was eroded	Good	Rapid recovery. Good stump.
June 23, 1874	12	George R—	9	Distortion and deformity from infantile paralysis of leg	Lateral flaps; patella left	Good	Good stump.
Sept. 13, 1872	13	Walter G—	20	Useless and ulcerated stump after amputation for injury 9 years before	Lateral flaps; patella left	Good	Wound sloughed and gaped a little and exposed inner condyle, from which the cartilage subsequently came away. A good stump ensued.

July 6, 1875	14	William P—	16	Old disease of the knee of eight years' standing; the limb wasted and flexed	Lateral flaps; patella, which was fixed to femur, left	Good	Very rapid recovery in three weeks. Excellent stump.
April 28, 1881	15	John G—	68	Epithelioma of the leg, with the tibia	Lateral flaps; patella left	Good	Some sloughing of stump, exposing bone, which necrosed. Good stump eventually.
Jan. 19, 1882	16	Miss —	13 wks.	Congenital malformation of leg; absent tibia	Lateral flaps; patella left	Good	Union in five days.
Feb. 28, 1882	17	George L—	30	Chronic ulcer and epithelioma involving tibia	Lateral flaps; patella left	Good	Rapid recovery.
June, 1883	18	Maria W—	30	Diseased and displaced knee, with paralysis of leg	Lateral flaps; tibia separated by saw from one condyle of femur; patella left	Good	Rapid recovery. Good stump.
Oct. 13, 1868	19	Harriett N—	47	Gangrene of leg from embolus of popliteal artery and endocarditis	Long anterior flap and short posterior flap; patella left	Stump healed	Died in fifth week from heart disease.

Traumatic Cases.

Mar. 12, 1872	1	Henry B—	21	Gangrene of leg following obstructed external iliac artery, the result of a fractured pelvis 26 days previously	Antero-posterior flaps; patella removed	Died on 11th day	Sloughing of flaps and pyæmia.
May 22, 1874	2	William M—	28	Gangrene of leg following ruptured femoral artery 17 days before	Lateral flaps; patella left	Good	Excellent stump.

Date of operation.	No. of case.	Name of patient.	Age.	Disease or injury.	Nature of operation.	Result.	Remarks.
Feb. 10, 1874	3	Henry T—	14	Compound fracture of leg	Primary amputation; lateral flaps; patella left	Good	Capital stump.
Feb. 10, 1874	4	William E—	18	Crushed foot and leg	Primary amputation; lateral flaps; patella left	Good	Small slough on posterior flap from pressure of splint. Excellent stump.
Aug. 22, 1875	5	Catherine A—	61	Compound comminuted fracture of leg	Secondary amputation; lateral flaps; patella left	Died on 11th day	Sank.
Jan. 26, 1876	6	James G—	44	Compound fracture of leg	Secondary amputation; lateral flaps; patella left	Good	Small slough the size of half a shilling on one flap.
April 8, 1879	7	James L—	68	Compound fracture of leg, &c.	Secondary amputation; lateral flaps; patella left	Sank on 8th day	—
Feb. 21, 1878	8	Thomas B—	60	Compound fracture of leg 15 days previously	Secondary amputation; lateral flaps; patella left	Good	Good stump after 16 weeks.
May 23, 1877	9	Phœbe C—	8	Crushed and lacerated leg, with much loss of blood before operation	Primary amputation; lateral flaps; patella left	Died in 3rd week	Sloughing of stump and pyæmia.
Dec. 5, 1882	10	Stephen G—	52	Compound fracture of leg	Secondary amputation; lateral flaps; patella left	Died on 17th day	Septicæmia.
Feb. 13, 1883	11	William D—	30	Compound fracture of leg 19 days previously	Secondary amputation; lateral flaps; patella left	Died on 33rd day	Stump healed all except one sinus. On the 33rd day there was secondary hæmorrhage from the femoral artery, and death.

Analysis of cases.—Thirty cases have been tabulated, and of these, nineteen were amputations performed for disease or for reasons of expediency, and eleven for injury. In the group of nineteen *amputations for disease*, one patient only died from the operation (Case 8), a man aged thirty-two, suffering with epithelial cancer involving the tibia. He sank on the fourth day from kidney disease. Of the eleven *traumatic* cases six died (Nos. 1, 5, 7, 9, 10, 11), and of these it is fair to say that the operation simply failed to save life, since it was performed in Case 1 for gangrene due to obstruction of the external iliac artery, the result of over-stretching of the vessel by a displaced fragment of a broken pelvis; in Case 9 as a primary amputation of a limb, crushed by the passage of a tramcar over it, in a child aged eight who had lost much blood before the operation; and in Cases 5, 8, and 10 as a secondary amputation for compound fracture of the leg in a woman aged sixty-one, and in men respectively fifty-two and sixty-eight years of age. Case 11 was remarkable, since death took place on the thirty-third day from secondary hæmorrhage, the result of an abscess in the extremity of the popliteal artery, which had been twisted at the time of operation. The patient was a man aged thirty, for whom a secondary amputation had been performed for compound fracture. The stump had healed, with the exception of one sinus, which evidently led down to the popliteal artery. I give an account of the preparation obtained after death from the pen of my friend, Mr. John Poland: "The stump presented on either side an almost level granulating surface, and on the posterior aspect another granulating surface running vertically upwards. At the upper end of this there was a sinus leading into a small abscess cavity in the position of the end of the popliteal artery. From this sinus a good-sized stream of water flowed when the common iliac was injected by means of a syringe. The walls of the abscess cavity were composed of soft shreddy slough, and there was an entire absence of granulation tissue. About the middle of

this abscess the divided end of the popliteal artery lay, presenting a most interesting condition. Suppuration had taken place between its middle and inner coats, burrowing upwards in such a manner as to completely separate the two for a distance of an inch and a half, so that the internal coat lay like a cast in the middle of the tube, and looked not unlike a coagulum. The upper part of this inner coat was thin and papery, and the upper limit of its separation from the outer coats abrupt and well defined. The lower three fourths of an inch, shaggy, softened, and sloughing, lay loose in the cavity. Above this presented the opening into the interior of the tube-like inner coat, which for a distance of an eighth of an inch contained the remains of some broken-up adherent coagulum. It was from this orifice that the hæmorrhage had taken place into the suppurating cavity and sinus. Corresponding to the whole length of the suppuration between the coats, the outer coats were found to be thickened by inflammatory material to double their normal size, and this condition extended upwards for a quarter of an inch above the upper limit of separation of the coats. Above this all the arterial coats appeared to be healthy. Below, the outer coats were continuous with that lining the abscess cavity and ended indistinguishably in it.

"The femoral vein was plugged for a distance of five inches with firm adherent clot. The suppuration between the coats of the popliteal artery being directly continuous with the cavity below, was clearly due to an inflammatory affection extending from the latter to the coats of the vessel, setting up a diffused suppurative arteritis. This inflammatory process is of an exceedingly unhealthy character, as shown by the sloughy condition of the wound and twisted end of the artery.

"I believe that all cases of secondary hæmorrhage at the present day will be found to be directly traceable to this particular inflammatory condition of the wound implicating the arterial coats. That this is a so-called septic form of inflammation I am not inclined to believe, but

rather that it may be dependent, to a very great extent, on some particular tendency of the patient."

Remarks.—Upon the whole, the operation, with respect to its dangers, must be regarded with favour. Of nineteen cases of amputation for disease, one only, or about 5 per cent., died; and of the traumatic cases about 50 per cent. were fatal, whilst the causes of death in these fatal cases were, in all the examples tabulated, due rather to general causes than to any condition which can be directly attributed to the operation itself.

On sloughing of the flaps.—With respect to this question, as determined by the cases tabulated, sloughing took place in four of the nineteen cases of amputations for disease.

In Case 2 the slough was enough to expose a portion of one condyle of the femur from which the cartilage exfoliated.

In Case 7, that of a man aged fifty, who had epithelioma of the leg involving the tibia, amputation was performed with an anterior flap of median length and a posterior flap. A small slough on the anterior flap took place, but with no detriment to the patient.

In Case 13, amputation was performed in a man aged seventy, for a useless and ulcerated stump of the leg after an operation done nine years previously for some injury. Lateral flaps, after Stephen Smith's method, were adopted. Some sloughing of one flap took place, and exposed the inner condyle from which the cartilage exfoliated; but a good stump was subsequently secured.

In Case 15, that of a man aged sixty-two, with epithelioma of the skin and tibia, lateral flaps were also made. One of these sloughed and exposed the corresponding condyle of the femur, which underwent superficial necrosis. But in this case, as in the preceding, a good stump was secured.

Sloughing also followed in two of the five successful amputations for injury; but in both it was very limited,

and proved in no way detrimental to the usefulness of the stump. In one of these—Case 4—a primary amputation was undertaken in a boy aged fifteen, for crushed foot and leg; lateral flaps were made, and a small slough formed in the posterior angle of one of the flaps from the pressure of a splint.

In Case 6, one of secondary amputation for compound fracture in a woman aged forty-four, a slough, the size of half a shilling, took place in one of the flaps, but a good stump resulted.

It occurred also in two of the fatal cases, but was in no way to be attributed to the operation (Cases 1 and 9).

Regarding the sloughing process with respect to the nature of the operation performed, it may be stated that of three cases in which the long anterior flap was employed (two for disease and one for injury), there was sloughing to a degree in two. In one of these, however (Case 1 in the table) gangrene had already resulted from plugging of the external iliac artery.

In six of the cases (all pathological) an anterior flap was made of medium length—about three inches and a half—extending from the posterior margin of the condyles downwards to a point an inch below the tubercle of the tibia. This was combined with a posterior flap of about two inches reaching down to the level of the tubercle of the tibia. In these six cases there was sloughing in one only (Case 7), in which, however, the process was very limited.

In the remaining twenty-one cases the operation was performed with lateral flaps, after the method of Stephen Smith. Eleven of these were amputations for disease. In two (Cases 13 and 15) sloughing occurred; whilst ten were traumatic cases, in three of which (Cases 4, 6, and 9) there was sloughing, but in the last case (9) the sloughing process was unconnected with the form of amputation.

Out of the whole thirty cases sloughing to a degree took place in eight. But if we eliminate Cases 1 and 9 in the traumatic table, in which the sloughing process

had no relation to the operation itself, the number is reduced to six, or to one in every five cases.

In none of the successful cases did sloughing take place to any extent, and it never materially interfered with the subsequent value of the stump.

In the cases in which there was no sloughing an excellent stump was obtained. No trouble was ever experienced from the articular cartilage over the condyles of the femur during the healing process, and when the stump had healed, the soft parts moved freely and loosely over the end of the bone. The cicatrix in all the cases was placed well behind the femur (*vide* Fig. 3, p. 177).

In all but the first three operations the patella was preserved; the removal of this bone I found to be quite unnecessary.

Patients after this operation are usually able to bear any amount of pressure upon the stump, and they can walk with greater facility than can patients after any form of amputation through the thigh. This result is probably due to the fact that the attachments of the muscles of the thigh, and particularly of the adductors, are less interfered with than they are in supracondyloid amputations.

For my own part, I know of no great operation which is followed by less shock, which repairs so rapidly and with so little constitutional disturbance, which forms a better and more useful stump, and which enables a patient to walk so well with an artificial leg.

THE OPERATION.—Three different methods have been advocated,—the long anterior flap of Pollock, the lateral hooded flaps of Stephen Smith, and the lateral flaps of Pick.

The first operation, as described by Pollock,[1] is as follows: "I make it a rule to feel for the interval between the edges of the condyle and head of the tibia, and to commence my incision at that point, and immediately behind the edge of the hamstring muscle, as it crosses that

[1] 'Med.-Chir. Trans.,' vol. liii, 1870.

space. I take especial care never to commence my incision higher than the margin of the condyle. The incision should be carried perpendicularly downwards on the side of the leg till nearly five inches below the lower edge of the patella, then gradually brought across the front of the leg, and when crossing the tibia should be quite five inches below the patella; then carried up the inner side to a point corresponding exactly to that from which the incision commenced. If the knife is introduced higher up than at the point mentioned, the incision will not only be longer than requisite, but the blood-vessels on each side, which pass from behind forwards, are unnecessarily divided at the base of the flap, and consequently its arterial supply is diminished by so much, and sloughing or ulceration of some portion of its extremity rendered more probable. I usually make the posterior flap by cutting from without inwards; it should not be too short, and should consist merely of integument. As soon as the flaps are completed all the structures round the joint should be divided at a right angle with the limb."

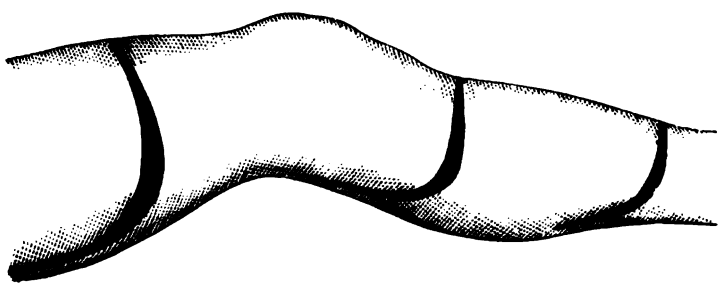
Pick's operation is described as follows:[1] "An incision was commenced at the upper border of the patella, and carried down the middle line of the limb as low as the tubercle of the tibia; it was then curved outwards over the outer side of the leg to the back, and carried upwards along the middle line to a point corresponding to the commencement of the incision on the front of the leg. A similar incision was carried round the inner side of the leg, and thus two somewhat quadrilateral flaps with rounded corners consisting only of skin and subcutaneous tissue were mapped out. The lowest point of these flaps was about an inch and a half below the level of the tubercle of the tibia. They were dissected up as high as the articulation, the patella was removed, and the various structures around the joint divided by a circular sweep of the knife." Pick claims for his operation the following advantages: that better drainage is secured than by that of the long

[1] 'Proceedings of Medical Society of London,' vol. vii, 1884, p. 134.

anterior flap; that the flaps are less liable to slough; and that the cicatrix is placed in the intercondyloid notch between the two prominent condyles of the femur, and is not therefore subjected to any direct pressure from the artificial limb. These claims, when compared with Pollock's long anterior flap are just.

Mr. Stephen Smith's amputation is described as follows:—"The incision is commenced about an inch below the tubercle of the tibia (Fig. 1), and carried down-

FIG. 1.



ward and forward over the most prominent part of the side of the leg, until it reaches the under surface, when it is curved towards the median line. When that point is reached, it is continued directly upward to the centre of the articulation. A second incision begins at the same point as the first, and pursues a similar direction upon the opposite side of the leg, and meets it in the median line in the posterior part. The line of incision upon one side is seen in Fig. 1. The following points," adds Stephen Smith, "should be remembered, viz. the incisions should incline moderately forwards down to the curve of the side of the leg, to secure ample covering for the condyles; and

that upon the internal aspect should have additional fulness for the purpose of ensuring sufficient flap for the internal condyle of the femur, which is longer and larger than the external. In the dissection the skin, fascia, and cellular tissue are raised and the ligamentum patellæ is divided, allowing the patella to remain. The appearance of the flaps immediately after disarticulation is seen in Fig. 2. It will be noticed that the extremity of the femur

FIG. 2.

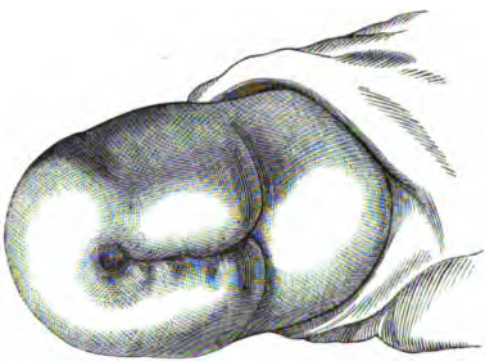
Appearance of flaps immediately after disarticulation.

is already completely covered, and the line of union of the flaps will be between the condyles and over the intracondyloid notch. When cicatrisation is complete the cicatrix sinks into this notch and disappears from the face of the stump, and offers no point of contact with the artificial appliance. The appearance of the stump on recovery is given in Fig. 3. In the process of repair, it will be found

that the drainage is so perfect that all the anterior portion of the wound remains dry and frequently heals by immediate union.

"This method of amputation need not be limited to the knee. The advantages of drainage, and the removal of the cicatrix from the face of the stump to its posterior part, equally adapt it to amputation in the leg or thigh. I have frequently amputated at both of these points by this method and obtained the most satisfactory results. The wound heals with remarkable rapidity, and the final perfection of the stump leaves all that can be desired. In Fig. 1 the line of incisions is given in amputation of the leg and thigh by this method. The incision on the

FIG. 3.



Posterior view of stump.

posterior part of the leg should extend upwards to the point where the bone is to be sawn through, and there the muscles are divided circularly."[1]

[1] 'American Journal of Medical Sciences,' January, 1870.

I have described this operation in Dr. Stephen Smith's own words, and illustrated it with copies of his original woodcuts, Figs. 1 and 2, in order to do his operation full justice, and that there should be no misunderstanding as to his method. I endorse all his remarks fully, and would urge the application of his method of operating at the knee to the leg as strongly as I can. Indeed, I may say that his operation upon the leg, with but slight modifications, has been practised at Guy's Hospital for more than forty years, although it is difficult to discover with whom it originated. My friend, colleague, and teacher, Mr. E. Cock, whose memory goes back to Sir A. Cooper, is unable to say when it was introduced, and I am rather disposed to think that the line of incision was Mr. Cock's. That excellent surgeon, however, has always included the muscles of the leg in his flaps, and, I am bound to add, with a good result. This practice of including muscle in the flaps has not, however, been adopted by all his colleagues or followers. In thin subjects it has been the rule, in others it has been the exception. The muscles are then divided by a circular cut. The stumps that result from this form of amputations in the leg, as in the knee, are perfect, and are certainly better than those obtained by the majority of other forms of amputation. In an examination at a certain university, where a candidate performed the operation, it was condemned, the examiners, having regarded it as a fancy measure, being unaware that it had been extensively practised at Guy's Hospital, and that it had also been for years before the profession in the pages of a student's text-book which has passed through many editions. I repeat here the two drawings which have illustrated the operation since 1872 (Figs. 4, 5). This ignorance of its value is to be regretted, and if these lines will help its progress towards more general adoption their author will be satisfied.

Of the three methods advocated for amputation at the knee, that of Stephen Smith is greatly to be preferred,

since it provides a better covering for the condyles of the femur than is obtained by any other method, and the

FIG. 4.

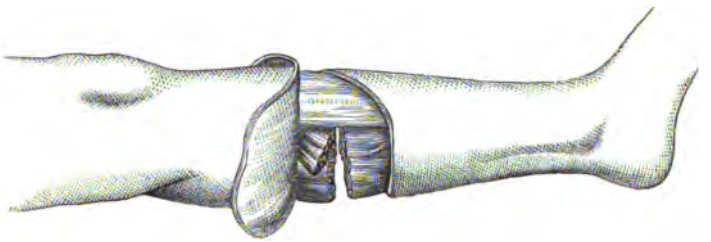
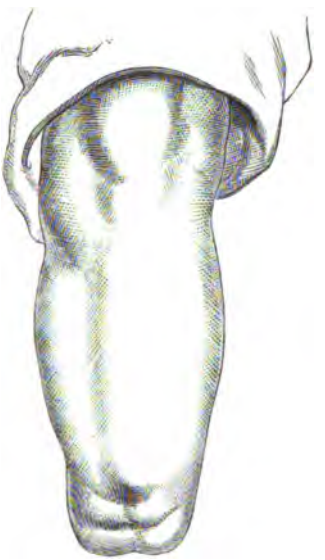


FIG. 5.



flaps are far less prone to slough than in the long five-inch anterior flaps advocated by Pollock. This view is

supported by the fact that of my own twenty-one cases, in only four, or in one out of every five cases, could it be said there was any sloughing, and in all of these the process was of a limited extent; whereas, out of Mr. Pollock's five cases in which this operation was performed there was sloughing in three; and out of twenty-nine other cases tabulated by Pick from the St. George's Hospital records, sloughing occurred in sixteen, or, taking the whole number of cases in which a long flap was made as thirty-four, sloughing followed in nineteen, that is, in 55 per cent., or more than half.

The method advocated is likewise to be preferred to Mr. Pick's operation, from the fact that in the former the incision is commenced one inch below the patella, and, as a consequence, the cicatrix is eventually placed entirely behind the condyles and out of harm's way; whereas, in Pick's operation the incision starts from a point above the patella and the cicatrix lies in the intercondyloid notch.

With Stephen Smith's flaps, moreover, there is no place for bagging of fluids after the operation; for, with the patient on his back, and with the femur horizontal, the edges of the flaps when brought together present downwards towards the plane of the body, and consequently the stump is in the best position for drainage. The flaps at the same time form a complete hood to the condyles.

In performing this operation I have, on four occasions, after completing the skin flaps, and—with the knee flexed upon the femur—making an incision along the anterior border of the head of the tibia, so as to divide the coronary ligaments, and expose the joint, found the semilunar cartilages closely encircling the condyles of the femur. So tightly did they do so that on the two occasions on which I removed them they had to be dissected from their position. In the two other cases they were left *in situ*, to the great advantage of the patients. Indeed, I would suggest that this latter practice should be the one followed where it can be, for by this means the upper part of the synovial capsule is held down firmly to

the condyles of the femur, and thus all the soft parts are kept well in place.

Since writing the above I find Dr. Brinton, so early as 1872, advocated this practice[1] in the following words: "I divide the coronary ligaments so as to allow the semilunar cartilages to remain upon the articular end of the femur and in the stump. By thus leaving them in position I have a cap fitted upon the end of the femur, which preserves all the fascial relations, eventually prevents retraction and guards against the projection of the condyles. I insist somewhat strongly upon this retention of the semilunar cartilages, since I regard it as having an important bearing upon the future wellbeing of the stump."

It is more than probable that this displacement of the interarticular fibro-cartilages may take place under other circumstances as by some accidental rupturing of the coronary ligaments, and if so, some of the cases of injury to the knee now registered under the title of internal derangement may be explained.

There is but little bleeding in this operation, and, with the exception of the popliteal and two superior articular arteries, there are none to twist. The popliteal vein had better be tied by a carbolised gut ligature. The condyloid origins of the gastrocnemius muscle had better be removed.

By way of conclusion, the advantages of this form of operation over amputation through the thigh may be stated as follows:—

1. The lessened shock of operation.

2. The lessened section of tissues and the non-exposure of the muscular interspaces of the thigh.

3. The escape from the necessity of sawing the femur, with its attendant risks.

4. The preservation of the attachments of the thigh muscles, and consequently the greater mobility of the stump.

[1] 'Philadelphia Medical Times,' December 28th, 1872.

5. And last but not least, the useful character of the resulting stump.

FIG. 6.

Artificial limb adapted to stump after operation.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' New Series, vol. ii, p. 27.)

ON THE INCREASE

IN NUMBER OF

WHITE CORPUSCLES IN THE BLOOD IN INFLAMMATION,

ESPECIALLY IN THOSE CASES ACCOMPANIED BY SUPPURATION.

BY

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(COMMUNICATED BY DR. RINGER, F.R.S.)

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DR. RINGER, in speaking to me of inflammation, mentioned the fact that although various writers had observed and recorded the increase of white blood-corpuscles in this condition, still this increase had never apparently been observed in a systematic manner in a series of cases, and he suggested that I should make the following observations.

Before doing so, however, I looked up the previous works on this point, and I found that the observers mentioned below had recorded this increase. Unfortunately, they have not all used the same method of counting, some of them having used diluted and others undiluted blood, while some have given their results per cubic millimetre, and others in the relative numbers of the corpuscles only.

Piorry in 1837[1] concluded from experiments on coagulation of the blood in pneumonia that the white blood-corpuscles were increased in that disease. Virchow[2] states that he has found an increased number of white blood-corpuscles in severe inflammations, especially in pneumonia, the typhoid state, and puerperal fever, and Nasse is quoted as having corroborated this statement as far as some cases of pneumonia are concerned. Concerning the chronic inflammatory conditions, it is stated[3] that Nasse has found this increase in phthisis, and Virchow and Gulliver have also recorded it in chronic diseases accompanied by hectic. But Malassez,[4] in 1873, published estimations of conclusions from a series of cases which are so interesting that I venture to quote them rather more fully. In looking over the results recorded by Malassez, however, it must be remembered that he takes 8000 white blood-corpuscles and 5,000,000 red blood-corpuscles as the normal number of corpuscles in a cubic millimetre of blood, which gives the relative number as 1 white to 625 red blood-corpuscles. He first quotes four cases of facial erysipelas without any complication, and gives estimations made during (1) the continuance of the rash, (2) during convalescence, (3) after complete recovery.

Case 1.—Woman, æt. 53.

Estimations during the eruption . .	1 W.B.C. to 333 R.B.C.
„ „ „ . .	1 W.B.C. to 533 R.B.C.
„ „ „ . .	1 W.B.C. to 535 R.B.C.
„ „ convalescence . .	1 W.B.C. to 986 R.B.C.
„ after recovery . . .	1 W.B.C. to 644 R.B.C.
„ „ . . .	1 W.B.C. to 525 R.B.C.

Case 2.—Woman, æt. 32.

Estimation during the eruption . .	1 W.B.C. to 480 R.B.C.
„ „ convalescence . .	1 W.B.C. to 895 R.B.C.
„ after recovery . . .	1 W.B.C. to 488 R.B.C.

[1] 'Traité des Altérations du Sang.'

[2] 'Gesammelte Abhandlungen zur wissens. Med.,' 1856, p. 180.

[3] Loc. cit.

[4] 'Bulletin de la Société Anatomique,' 1873, p. 141.

The other two cases are not complete, but they confirm the above figures.

From these cases Malassez concludes that there is :

1. An increase of white blood-corpuscles during the eruption of erysipelas.

2. A decrease of white blood-corpuscles when the eruption disappears.

3. A return of the white blood-corpuscles to their normal number during the week following convalescence.

But the above apparent increase is only relative, because in Case 1, during the eruption, the red blood-corpuscles fell from 4,100,000 per cubic millimetre to 3,600,000, although, when the eruption faded, the white blood-corpuscles were actually decreased, but not so much as the proportion indicates, because at that time the number of red blood-corpuscles only amounted to 4,000,000 per cubic millimetre.

In Case 2 also the red blood-corpuscles rose at the end of the disease from 3,700,000 to 4,100,000 per cubic millimetre.

Malassez next quotes a case of facial erysipelas followed by suppuration near the sterno-mastoid muscle in which, during the eruption, there was 1 white blood-corpuscle to 400 red blood-corpuscles; when the abscess was forming 1 white blood-corpuscle to 342 red blood-corpuscles; when the abscess had increased in size 1 white blood-corpuscle to 295 red blood-corpuscles; after the pus was removed 1 white blood-corpuscle to 345 red blood-corpuscles, 1 white blood-corpuscle to 385 red blood-corpuscles.

So that in this case of facial erysipelas complicated with suppuration there was no greater increase in the number of the white blood-corpuscles than in an ordinary case of erysipelas, so long as it alone existed, but a further increase was at once observed when suppuration commenced, "and this only ceased when the pus escaped."

Two cases of erysipelas are then related in subjects suffering from chronic enlargement of the cervical glands, which confirm the above observations.

In the same article it is stated that Vulpian and Troisier had examined three cases of erysipelas, and although these experiments were made with undiluted blood they found that the white blood-corpuscles were increased in each case, and in one of them, in which an abscess was opened at the same time that the erysipelas was cured, the number of white blood-corpuscles in each field of the microscope fell from 25 to 10.

Liouville and Béhier have also observed the increase of white blood-corpuscles in erysipelas, and Berger, quoting from Klebs, says that the white blood-corpuscles are increased both in suppuration and in pneumonia.

Nicati and Tarchanoff[1] compare the increase in the number of white blood-corpuscles caused by severe and slight inflammations, and they show that the more severe the inflammation the greater is the increase in the number of the white blood-corpuscles. In comparing the number of white blood-corpuscles contained in the venous blood returning from an inflamed area with the number in the venous blood generally of the body of a rabbit, they found a large increase in the former and a relative increase in the latter.

English writers appear to doubt this increase, if the following passage from Erichsen's 'Surgery' may be taken as fairly expressing their views :—"As to the white corpuscles we know that they are present in augmented numbers in the vessels of the inflamed part; whether they are really more numerous in the blood in inflammation is doubted by Paget, Simon, and others."

The observations recorded by myself in this paper were made with a Gowers' hæmocytometer, as described in Quain's 'Dictionary of Medicine,' p. 561. But after some experience I found that practically it was quite sufficient to count the number of red blood-corpuscles in four squares of the cell instead of in ten as Gowers recommends, as this gave in the end the same average number of red blood-corpuscles per square as when the larger

[1] 'Archives de Physiologie normale et pathologique,' 1875, p. 514.

number of squares were counted, provided that the blood was thoroughly mixed with the diluting fluid. I have also in the following estimations, when counting the white corpuscles, slightly lowered the focus, and have then counted the white blood-corpuscles as dark coloured spots. This plan I have found to be easier, quicker, and quite as correct as that recommended by Gowers, in which the focus is slightly raised and then the white corpuscles counted as bright points.

The results are given in percentage number of red, and relative number of white, blood-corpuscles.

The average number of corpuscles contained in a cubic millimetre of blood is given by Dr. Gowers as 15,000 white and 5,000,000 red blood-corpuscles, which gives a proportion of 1 white blood-corpuscle to 333 red ones.

CASE 1.—Case of iliac abscess, elastic, but not fluctuating at commencement of observations.

Date. Feb.	Temp. a.m.	Temp. p.m.	Per cent. No. of R.C.	Relative No. of W.C.	
10	99·8°	101°	90	1 to 139	
12	100	99·4	93	1 to 202	
15	99·2	100	88	1 to 137	Average 1 to 160.
			92	1 to 138	
			90	1 to 139	
18	99·4	99·8	92	1 to 86	
Abscess increasing in size fluctuation can now be obtained.					
20	99·2	99	86	1 to 110	
21	98·6	100·6	86	1 to 86	
22	99·6	100·2	86	1 to 104	Average 1 to 101.
24	98·4	100	98	1 to 91	
25	99·6	100·4	86	1 to 124	
26	—	98	90	1 to 96	
Operation at 2.30 p.m. on Feb. 26, immediately after last observation.					
Large amount of pus escaped when abscess was opened.					
Free discharge of pus and serum in first 24 hours after incision.					
27	100·2	101·8	94	1 to 333	
28	97·8	98·6	90	1 to 270	
March 1	98	98·2	88	1 to 304	
2	—	98·2	98	1 to 245	

Date.	Temp. a.m.	Temp. p.m.	Per cent. No. of R.C.	Relative No. of W.C.	
March 3	—	98·6°	92	1 to 230	
4	—	99·2	96	1 to 105	Average 1 to 203.
Constipation present; bowels not relieved since operation. Not thoroughly opened until March 9th. Discharge much less.					
5	—	103	94	1 to 114	
6	99°	98·4	88	1 to 158	
7	103	102	84	1 to 161	
8	98·8	102·4	86	1 to 150	
10	—	99·4	92	1 to 184	
11	98·4	98·6	96	1 to 240	Average 1 to 223.
12	—	98·4	96	1 to 300	
13	98·6	99·4	90	1 to 250	
14	98·4	98·4	92	1 to 287	
15	99·6	101	94	1 to 213	
16	98·4	98	88	1 to 314	
17	98	98	90	1 to 287	
19	98·4	97·8	92	1 to 286	Average 1 to 252.
20	—	98·2	94	1 to 313	
21	98	98·2	94	1 to 335	
22	97·8	98	98	1 to 272	
23	98·4	98·2	96	1 to 320	
24	98·4	98·2	90	1 to 264	
26	98·4	97·8	94	1 to 361	
27	—	97·8	94	1 to 408	
March 27th.—Drainage changed for one half the original diameter and length. Discharge almost stopped.					
28	98·4	97	96	1 to 300	Average 1 to 358.
31	99	98·4	96	1 to 320	
April 2	98	97	90	1 to 450	
4	—	97·4	96	1 to 400	
Drainage-tube 2 inches long. Discharge nil.					
8	—	98	90	1 to 346	
22	—	97·4	88	1 to 315	

Patient got up on 19th April. The drainage-tube was removed, and the wound had completely closed by the 21st, the patient leaving the hospital on April 22nd.

This case is one of iliac abscess coming on about four months after a confinement. On admission to Univer-

sity College Hospital there was an elastic, tender, and painful swelling in the left iliac fossa reaching two inches above Poupart's ligament. The glands in the groin were enlarged although the skin was not red over the swelling. Observations were made on ten days between February 10th and 26th. During the first half of this period the average relative number of white blood-corpuscles was 1 to 160, and during the last half, when there was fluctuation in the swelling, 1 to 101, which shows that there was a decided tendency to increase. All this time the swelling was increasing in size, and on February 26th the white blood-corpuscles had reached the relative number of 1 to 96.

On the following day (February 27th) the abscess was opened and there was an immediate fall to 1 to 383. After this the white blood-corpuscles increased in number, the average of daily estimations made from February 28th to March 6th (inclusive) being 1 to 203; from March 5th to March 10th there was slight fever, which was supposed to be caused by constipation, but it is quite possible that this rise in temperature was due to a slight increase of inflammation in the walls of the abscess; and, if this was the cause, the rise in the number of the white blood-corpuscles mentioned above would be accounted for. After March 6th the white blood-corpuscles showed a gradual decrease in number, the averages obtained from two periods of seven days each being 1 to 223 and 1 to 252. By March 23rd, the abscess cavity had almost entirely filled with granulation tissue, the discharge had almost ceased, and the white blood-corpuscles had become normal in number, viz. 1 to 320.

From this date to April 22nd, when the wound had been closed fourteen days, the white blood-corpuscles continued to have a normal relation to the red corpuscles, the average being 1 to 358.

CASE 2.—Case of pelvic cellulitis. Small swelling in right iliac region, large one in pelvis.

Date.	Temp.		Per cent. No.	Relative No.	
Feb.	a.m.	p.m.	of R.C.	of W.C.	
18	99°	100·4°	90	1 to 124	
21	99·8	101·6	92	1 to 153	
22	100·6	102·4	82	1 to 153	Average 1 to 148.
24	100·4	102·6	96	1 to 147	
26	100	103·4	82	1 to 167	
March					
1	100·2	103·6	96	1 to 168	
Temperature from March 1st to 10th varied from 98·8° a.m. to 103·4° p.m.					
10	101	103	86	1 to 159	Average 1 to 172.
14	101	103·8	94	1 to 204	
15	101	102·4	86	1 to 187	
16	102·2	103·4	80	1 to 166	
17	—	102·4	76	1 to 126	
19	100·8	102	70	1 to 112	
20	100·8	102	80	1 to 129	Average 1 to 150.
22	99·4	100	74	1 to 142	
23	99·2	99·6	88	1 to 244	
24	99·6	99·4	76	1 to 165	
26	—	—	74	1 to 154	
Observations from March 26th to May 4th were lost, but they showed a large and persistent increase of white blood-corpuscles. Patient is improving, but in above interval temp. varied from 99·8° to 103°.					Average 1 to 158.
May					
4	100	101·2	82	1 to 186	
5	—	101	92	1 to 135	
6	100·4	101	82	1 to 151	
7	99·4	101·2	82	1 to 128	
8	99·8	101·2	84	1 to 168	
9	98·6	100·2	82	1 to 164	Average 1 to 167.
10	100	100·2	92	1 to 191	
11	100	101·2	86	1 to 186	
13	99·8	100·4	80	1 to 166	

Improvement continued, but at this date it suddenly increased.

On May 17th patient got up after 18 weeks in bed, and left the hospital on May 22nd.

Date.	Temp. a.m.	Temp. p.m.	Per cent. No. of R.C.	Relative No. of W.C.	
May					
15	—	99°	—	1 to 250	Average 1 to 296.
17	98·8°	99·4	—	1 to 293	
19	98·6	99·2	—	1 to 366	
21	—	100	—	1 to 275	

This is another case of pelvic cellulitis, accompanied by well-marked fever and increase in the number of the white blood-corpuscles. The cellulitis came on after an abortion, and when the observations were commenced there was a large tumour in the pelvis, which could be felt both per vaginam and by abdominal palpation. The temperature in this case varied between 99° a.m. and 103·8° p.m.

The relative number of white blood-corpuscles from February 18th to May 13th was as under, each number given being the average of five observations made on separate days.

1	1 to 148.
2	1 to 172.
3	1 to 150.
4	1 to 158.
5	1 to 167.

During the above period there was abdominal pain, tenderness, loss of appetite, flesh, and strength, but on May 15th the temperature became normal and the symptoms disappeared; during the next few days the patient rapidly became convalescent, and it was found that the swelling in the pelvis was certainly smaller.

On the same date (May 15th) the relative number of white corpuscles decreased to 1 to 250, and they continued to decrease until, on May 19th, there was only 1 white to 366 red blood-corpuscles, the patient leaving the hospital cured on May 22nd.

On looking at this case and observing the sudden fall of temperature, accompanied by loss of symptoms and dimi-

nution in the size of the swelling, we may infer, with the physician who had charge of the case, that an abscess had discharged itself into the bowel, an opinion that he formed without knowing that the white blood-corpuscles had been counted, and we may also presume that the diminution in the number of the white blood-corpuscles, which occurred at the same time, was due to this escape of pus, and that it corresponds with the diminution seen to occur in Case No. 1 when the abscess was opened.

Case 3.—Case of suppurating white leg. No observations until there was distinct fluctuation.

Date.	Temp.		Per cent. No.	Relative No.	
March	a.m.	p.m.	of R.C.	of W.C.	
2	99°	103·8°	68	1 to 145	
Abscess opened antiseptically; about 5 ounces of blood-stained pus escaped from beneath the soleus.					
3	100·2	102·6	74	1 to 133	Average 1 to 143.
4	100	102	90	1 to 122	
5	98·2	101·2	81	1 to 175	
Drainage not altogether perfect, but wound is granulating. Wound quite superficial. Patient to leave hospital on March 23rd.					
22	98·4	98·2	88	1 to 338	

The whole of the right leg was swollen, œdematous, and tender, with distinct redness over the centre of the calf where deep fluctuation could be obtained.

The temperature ranged, as is shown in the above table, from 99° a.m. to 103·8° p.m.

Before the pus was evacuated there was one white blood-corpuscle to 145 red ones, while the average for three days immediately following its evacuation was one to 143. During these days there was a fair amount of fever, the highest point reached during this time being 102·6°; but eventually, when the temperature became normal, the number of white blood-corpuscles became normal also.

This case appears to confirm what we have seen in part of Case 1, in which after the abscess had been opened, there

was a slight rise of temperature accompanied by an increase in the number of white blood-corpuscles; but there was an important difference in the two cases, because while Case 1 was perfectly sweet, Case 3 at this time was slightly fœtid and badly drained.

Case 4.—Case of double suppurative tonsillitis. First observation on fifth day of illness.

Date.	Temp.		Per cent. No.	Relative No.
May	a.m.	p.m.	of R.C.	of W.C.
22	99·9°	102°	98	1 to 326
23	99·8	101·4	96	1 to 287
Left tonsil discharged pus at 8 a.m. on May 24th. Observation made at 7 a.m.				
24	98·4	102·2	98	1 to 204
Right tonsil discharged pus at 7 a.m. on May 25th. Observation made at 8 a.m.				
25	101	102·2	96	1 to 369
26	98·8	98·8	92	1 to 270
28	98·2	98	100	1 to 500
Patient discharged well on May 28th. No treatment used in course of case except ice to suck.				

This case shows a slight but gradual increase in the number of white blood-corpuscles while the abscesses were forming, and this continued until both had discharged their contents, when the number of white blood-corpuscles at once fell to normal; the case also apparently confirms that which we may infer from the previous ones, viz. that the increase in the number of white blood-corpuscles is less in cases in which there are small, than in those in which there are large, abscesses.

Thus in Case 1, pelvic abscess, the highest average was 1 to 101; in Case 2, small pelvic abscess, the highest average was 1 to 150; in Case 3, small abscess in calf, the highest number was 1 to 145; while in Case 4, suppurative tonsillitis, the observation which shows the

largest number of white blood-corpuscles only gives 1 white to 204 red corpuscles.

The following estimations were made in two cases in which the actual cautery was applied for white swelling—one of the knee, the other of the shoulder.

Case 5.—Case of white swelling of knee treated by actual cautery. First observation made five hours after operation.

Date. Feb.	Temp. a.m.	Temp. p.m.	Per cent. No. of R.C.	Relative No. of W.C.	
19	98·4°	100·2°	92	1 to 129	Average 1 to 174.
21	98·4	101·4	94	1 to 220	
Marks left by cautery are now secreting pus; they are dressed with savin ointment.					
22	100·6	102	88	1 to 304	Average 1 to 321.
24	99·4	99·8	92	1 to 328	
26	99	99	96	1 to 332	

Case 6.—Case of strumous disease of the shoulder-joint treated by actual cautery. First observation four hours after operation. After operation the shoulder was kept at absolute rest. No irritation applied.

Date. Feb.	Temp. a.m.	Temp. p.m.	Per cent. No. of R.C.	Relative No. of W.C.	
19	98·4	98·2	98	1 to 220	Average 1 to 235.
20	98·4	99·8	98	1 to 294	
21	98·4	99·6	94	1 to 200	
Marks left by cautery discharging pus.					
22	97·8	99·2	96	1 to 308	Average 1 to 299.
24	98·4	98·4	96	1 to 286	
26	98·6	99	88	1 to 304	

In Case 5 there was rather more inflammation, as shown by the temperature which reached 100·2°, than there was in Case 6, where it only reached 99·8°, but in Case 5 there was also a much larger increase in the number of the white corpuscles after the operation than in Case 6, the average number in Case 5 being 1 to 174, while the average in Case 6 was 1 to 235.

Both cases were suppurating freely on the fourth day after

cauterisation, and there was no retention of discharge in either case. In both on the fourth day there was a marked decrease in the number of white blood-corpuscles.

In Case 5 they fell from 1 to 220, to 1 to 304, and in Case 6 they fell from 1 to 200, to 1 to 308.

After this day the averages show a near approach to the normal number of white blood-corpuscles, being in Case 5 1 to 321, and in Case 6 1 to 299.

I will next quote four cases of empyema in which observations were made both before and after the chest was drained.

Case 7.—Case of empyema; about ten ounces of seropurulent fluid removed by aspiration before admission to hospital. Aspiration repeated on April 27th and 29th, and about two ounces of pus removed each time, but on each occasion the needle became blocked by flaky lymph. Observations before aspiration showed a large increase of white corpuscles, but the figures have been lost.

Date. May	Temp. a.m.	Temp. p.m.	Per cent. No. of R.C.	Relative No. of W.C.	
4	100·2°	100°	82	1 to 132	
5	100·4	99·6	82	1 to 132	
Well-marked retraction of side.					
6	100·4	99·4	84	1 to 155	Average 1 to 168
7	99·4	99	88	1 to 275	
8	99·2	98·6	90	1 to 150	
9	99·8	98·4	82	1 to 132	
10	99·4	99	82	1 to 186	
Abscess pointing in 9th space, mid-axillary line.					
11	99·6	99·6	80	1 to 202	Average 1 to 177.
12	99·6	98·4	92	1 to 200	
13	99·8	97·4	83	1 to 148	
Abscess opened with Listerian precautions, drained, half inch of rib removed on the 13th, after observation. Dressed on the 14th.					
14	99·2	99·4	80	1 to 307	
15	99·2	99	84	1 to 350	
16	99	99·8	82	1 to 273	Average 1 to 315.
17	99·2	99	82	1 to 315	
19	98	99	86	1 to 330	
Dressing changed. Retraction much more marked.					

Date.	Temp. a.m.	Temp. p.m.	Per cent. No. of R.C.	Relative No. of W.C.	
May					
20	99·2°	98·6°	96	1 to 436	
21	98·6	99	98	1 to 300	
22	98	99·4	92	1 to 281	Average 1 to 325.
23	99	98·4	80	1 to 285	
24	98·2	99	94	1 to 313	
25	98	99	84	1 to 280	
26	99	99·4	84	1 to 233	
28	99·6	98·6	98	1 to 258	
30	99	99·8	88	1 to 338	Average 1 to 301.
Dressing changed May 31st.					
June					
1	99	99·8	88	1 to 400	
4	99	—	92	1 to 328	
6	99	99·2	88	1 to 366	
Dressing changed June 7th.					Average 1 to 332.
8	98·6	99·4	100	1 to 454	
11	99·6	—	94	1 to 180	
Wound completely closed on June 24th.					

This case occurred in a child, aged seven, in whom the signs of fluid in the right pleural cavity were well marked. On admission to the hospital, although about ten ounces of sero-purulent fluid had been previously withdrawn by aspiration, very little fluid could be obtained; repeated attempts to aspirate were made both with and without an anæsthetic, on each occasion the needle becoming blocked. Observations were made daily from May 4th to 13th as to the number of white corpuscles, and it was found that the first five days gave an average of 1 to 168, and the second five days an average of 1 to 177, results which are practically the same.

At the commencement of this series of observations, which was about fourteen days after admission, there was well-marked retraction of the whole of the right side of the chest. On May 9th it was noticed that the empyema was pointing, and on May 13th the chest was opened and drained, half an inch of rib being removed and a large amount of thick curdy pus being evacuated. The day

before the operation the relative amount of white to red corpuscles was 1 to 148; the day after, it had fallen to 1 to 307, the temperature remaining as before, just above normal, *i. e.* 99·2° a.m. and 99·4° p.m.

The average relative number of white corpuscles from May 14th to May 19th, which were the days immediately following the evacuation of the pus, was 1 to 315, which contrasts strongly with the averages before the operation. From May 19th until June 11th nineteen observations were made on separate days, and averages from these, divided into four periods, are given below:

1	1 to 315
2	1 to 325
3	1 to 301
4	1 to 332

figures which show a very close approach to the normal number.

On June 24th the patient left the hospital, the wound being completely closed.

CASE 8.—Case of empyema. Left side. Aspirated on May 7th. The average relative number of white blood-corpuscles was 1 to 187 before the chest was opened.

Date.	Temp. a.m.	Temp. p.m.	Per cent. No. of R.C.	Relative No. of W.C.	
May 7 ...	98·4° ...	101° ...	92 ...	1 to 242	Average 1 to 187.
8 ...	98·9 ...	— ...	98 ...	1 to 181	
(7.30 a.m.)					
8 ...	— ...	— ...	94 ...	1 to 140	
(3 p.m.)					
Abscess opened May 8th, drained, 50 ounces of pus removed.					
Dressed first time on 9th.					
9 ...	98·6 ...	99·8 ...	92 ...	1 to 200	Average 1 to 273.
10 ...	99·2 ...	99·6 ...	88 ...	1 to 258	
11 ...	100·2 ...	101·2 ...	90 ...	1 to 300	
12 ...	100·6 ...	101·4 ...	92 ...	1 to 328	
Dressed on 11th and 13th before estimations.					

Date.	Temp. a.m.	Temp. p.m.	Per cent. No. of R. C.	Relative No. of W.C.	
May 13	100·4°	101·4°	88	1 to 244	
14	101·8	101·4	94	1 to 276	
15	100·2	101·4	98	1 to 288	Average 1 to 278.
16	100·8	100·2	94	1 to 276	
17	100·2	100·2	92	1 to 287	
Dressed on 15th and 18th.					
19	99·8	99	94	1 to 392	
20	99·2	99·8	86	1 to 390	
Dressed on 21st.					Average 1 to 334.
22	—	99·6	92	1 to 306	
23	99·4	99·4	88	1 to 338	
24	99	99	88	1 to 244	
25	99	98·6	90	1 to 300	
26	99	99	90	1 to 409	
Dressed.					
28	99·6	98·2	84	1 to 350	Average 1 to 333.
30	99	99·2	88	1 to 314	
June					
1	99·6	99·2	70	1 to 291	
Dressed on June 3rd.					
4	99·6	100	96	1 to 400	
6	99·8	100	80	1 to 266	
8	99	99·2	90	1 to 409	Average 1 to 345.
11	98·8	—	86	1 to 307	

After the operation, however, although there was a considerable fall in the number of white blood-corpuscles, they did not fall to normal, the average obtained from nine daily observations, from May 9th to 17th, after the chest was opened and drained, being 1 to 273. Possibly this might be accounted for by the fact that in this case the patient was an adult, and as no bone was removed the chest did not drain satisfactorily at first.

After May 17th, however, the proportion of white to red corpuscles again fell, the averages for three periods of five days each being: 1 to 334, 1 to 333, 1 to 345, which figures show even a closer approach to the normal than was obtained in Case 7.

The observations were continued until June 11th, when the patient was lost sight of, as he left the hospital, although there was still a small sinus.

CASE 9.—Case of empyema in a child, following on pneumonia.

Date.	Temp. a.m.	Temp. p.m.	Per cent. No. of R.C.	Relative No. of W.C.	
June 30	98·4°	102°	70	1 to 125	Average 1 to 143.
„	—	—	84	1 to 140	
July 1	99·8	100	88	1 to 162	
„	—	—	82	1 to 146	

July 1st.—Free incision, 8 oz. of pus evacuated; half inch of rib removed. Next observation 8 hours after operation.

July 1	—	—	84	1 to 247	Average 1 to 360.
Immediately after operation.					
2	97·4	98	80	1 to 363	
4	98·4	98	80	1 to 488	
17	—	—	96	1 to 342	

This case corresponds almost exactly to Case 7, the proportions being as under.

Previous to operation the average of four estimations made on two days was 1 to 143.

After operation the average of four estimations made on four separate days was 1 to 360.

In this case also the fall in the number of white blood-corpuscles was observed the day after the operation, the number the day before being 1 to 146, and the number the day after being 1 to 363.

CASE 10.—Case of right empyema in a puerperal woman.

Date.	Temp. a.m.	Temp. p.m.	Per cent. No. of R.C.	Relative No. of W.C.	
Feb. 12	104	103	84	1 to 189	Average 1 to 193.
14	100	101	88	1 to 198	

Pus evacuated by incision; a large amount removed.

16	100·8	98·6	84	1 to 180	Average 1 to 187.
18	97·4	97·4	88	1 to 190	
19	98·8	99·6	92	1 to 192	

Death occurred on Feb. 19.

At post-mortem no further collection of pus was found.

CASE 10 is an empyema that occurred in a puerperal woman; it is only useful to show the increase of white blood-corpuscles in the presence of a collection of pus, the average relative number being 1 to 193.

The chest was opened four days before death, but this could hardly be expected to diminish the number of the white blood-corpuscles in a patient in such a condition, the average obtained from the estimation made after the operation being 1 to 187.

The observations made on these four cases of empyema corroborate those made on the cases of suppurative cellulitis, viz. that wherever there is a collection of pus there is an appreciable increase in the relative number of white blood-corpuscles, which falls as soon as this pus is evacuated.

The next three cases, 11, 12, and 13, are a series of observations made on phthisical patients with cavities and free expectoration; these all show a slight increase in white blood-corpuscles, as has been previously recorded by Nasse.

CASE 11.—*Phthisis* cavities over the whole of right lung, copious muco-purulent expectoration.

Date.	Temp.		Per cent. No. of R.C.	Relative No. of W.C.	
Feb.	a.m.	p.m.			
9	101·6°	103·6°	a.m. 92	1 to 350	Average 1 to 290.
			p.m. 94	1 to 297	
12	101·4	104	90	1 to 368	
18	100·6	103·6	86	1 to 298	
24	—	102	88	1 to 264	
March					
1	101	102·4	96	1 to 216	
4	99	103	74	1 to 237	

CASE 12.—*Phthisis* cavity at left apex, moderate amount of muco-purulent expectoration.

Date. Feb.	Temp. a.m.	Temp. p.m.	Per cent. No. of R.C.	Relative No. of W.C.	
8	100	101	89	1 to 225	Average 1 to 230
10	101	101·4	94	1 to 257	
19	100·6	100·2	88	1 to 208	

Case 13.—*Phthisis* cavities at both apices, muco-purulent expectoration. Spinal caries, psoas abscess, open, badly drained.

Date. Feb.	Temp. a.m.	Temp. p.m.	Per cent. No. of R.C.	Relative No. of W.C.	
10	102	103·4	84	1 to 171	Average 1 to 180.
12	100·2	102·2	86	1 to 153	
19	101	101·6	82	1 to 217	

Cases 11 and 12 are comparable to the case of iliac abscess which is recorded as Case 1.

This similarity is found in the fact that after the abscess in Case 1 was opened we have three patients in each of whom there is a cavity or cavities (although in different parts of the body) secreting pus in considerable quantities, the pus in each of the cases having comparatively free exit. It is also shown in the relative numbers of the white blood-corpuscles which are given below:

Averages from Case 1.—1 to 203, 1 to 223, 1 to 252.

Average from Case 11.—1 to 290.

Average from Case 12.—1 to 230.

Case 13 was, however, complicated by an open, badly-drained psoas abscess depending on spinal caries, and in this case we find that the relative number of white blood-corpuscles is increased not only above the normal, but also above the increase found in Cases 11 and 12, which are ordinary cases of phthisis; just as one might be led to expect from the observations made in Cases 3 and 8, in both of which there were abscess cavities badly drained.

In Case 13 the proportion of white and red corpuscles is 1 to 180; in 11 and 12 it is 1 to 290, and 1 to 230; while in Cases 3 and 8, whilst they were badly drained, it was 1 to 143, and one to 272.

Case 14.—Left pleurisy with serous effusion. Friction on April 27th. Serous fluid obtained on May 6th, which coagulated spontaneously. Movements diminished. Vocal fremitus absent. Breath-sounds absent. Heart's impulse on right of sternum.

Date. May	Temp. a.m.	Temp. p.m.	Per cent. No. of R.C.	Relative No. of W.C.	
5	100·6°	103·6°	90	1 to 264	Average 1 to 336.
6	101·2	103·6	82	1 to 292	
7	100·6	102·6	86	1 to 330	
8	100·6	103·2	92	1 to 306	
9	100·6	103·2	92	1 to 383	
10	102·6	103·4	88	1 to 400	
11	100·6	103	86	1 to 390	
12	101·4	103·4	98	1 to 326	

Heart's impulse still on right of sternum. Vocal fremitus at level of nipple, in anterior maxillary fold. No V.F. below this. Friction present.

13	100·8	102·2	88	1 to 400	Average 1 to 412.
14	100·4	101	92	1 to 353	
15	100·2	100·6	92	1 to 418	
16	99	100·8	86	1 to 390	
17	98	100·6	90	1 to 500	

No pulsation on right of sternum. Heart's apex beat felt in 4th space, left side, inside nipple. No friction. Vocal fremitus felt quite to the base.

19	98·6	98·4			
20	98·6	99	96	1 to 480	Average 1 to 421.
21	98·6	98·8	88	1 to 440	
22	98·6	99	96	1 to 436	
23	98·6	99	98	1 to 445	
24	98·6	98	90	1 to 321	
26	—	98·8	80	1 to 400	
30	98·4	99·4	86	1 to 430	

Patient was discharged on June 1st, with slight deficient resonance at left base, also with slight deficient movement and slight retraction of left side.

Here there was no increase in the number of the white blood-corpuscles whilst any fluid remained, the relative number being, from an average of eight observations, 1 to 336.

During convalescence, however, there was a decided diminution in their number, the averages being, during a period of five and seven days respectively, 1 to 412 and 1 to 421.

Here we have a case in which there was a considerable amount of inflammation, as is evident from the large amount of serous fluid which was present, and which was accompanied by high fever, the temperature varying from 100·6° a.m. to 103·6° p.m., but in which there was no increase in the number of white blood-corpuscles.

May we therefore infer from this that a special variety of inflammation is necessary in order to cause their increase?

Case 15.—Case of left serous pleurisy with effusion. Aspirated on May 4th after estimation. Fifty-two ounces of fluid withdrawn.

Date. May	Temp. a.m.	Temp. p.m.	Per cent. No. of R.C.	Relative No. of W.C.	
4	102°	103·4°	74	1 to 217?	
5	101·2	103	84	1 to 300	
6	101·2	102·6	80	1 to 307	Average 1 to 302.
7	101	103·2	84	1 to 300	
36 oz. of fluid withdrawn by the aspirator.					
8	101·6	103	92	1 to 383	
9	102	103·6	90	1 to 346	
10	101·2	102	84	1 to 466	Average 1 to 385.
11	100·6	102·2	90	1 to 409	
12	101	102·4	90	1 to 321	
Vocal fremitus felt at extreme base. Slight dulness at left base. Breath-sounds heard at extreme base. Slight cough and mucoid expectoration, streaked with blood.					
13	101	102·4	78	1 to 433	
14	101·2	102·4	86	1 to 430	
15	99·8	102·6	90	1 to 450	Average 1 to 440.
16	100·4	102·2	92	1 to 511	
17	101·6	101·8	86	1 to 377	
19	100·2	100·6	84	1 to 381	
20	99	100·8	82	1 to 410	
22	99·6	99·6	88	1 to 366	Average 1 to 378.
23	99·2	99·2	88	1 to 314	
24	99·2	99·8	84	1 to 420	

Date.	Temp. a.m.	Temp. p.m.	Per cent. No. of R.C.	Relative No. of W.C.	
May					
25	99·4°	99·8°	84	1 to 420	
26	98·2	99·2	80	1 to 500	
Patient got up on May 28th for the first time.					Average 1 to 447.
28	98·6	98·6	92	1 to 460	
30	98·2	98·8	90	1 to 409	
Patient discharged well on June 2nd.					

This case is similar in all its characters to the last, except that here aspiration was employed twice, and fifty-two ounces and thirty-six ounces of fluid were withdrawn on the respective occasions, while in Case 14 this was not done.

In this case the averages are, while fluid was present, 1 to 302 and 1 to 385; during convalescence, 1 to 440, 1 to 378, and 1 to 447.

These two cases form a very marked contrast with the cases of empyema previously spoken of, in which the white blood-corpuscles were very largely increased.

Case 16.—Case of lobar pneumonia, left base. Illness commenced on May 30th. Observations commenced on June 3rd, being fifth day of disease.

Date.	Temp. a.m.	Temp. p.m.	Per cent. No. of R.C.	Relative No. of W.C.	
June					
3	102	103·6	96	1 to 369	
4	101·2	105	92	1 to 242	Average 1 to 259.
5	103·4	98·6	80	1 to 166	
Crisis on early morning of eighth day.					
6	98·6	97·6	80	1 to 571	
7	98·2	98	82	1 to 410	
8	98·6	98·8	90	1 to 281	Average 1 to 344.
9	97·8	97·4	94	1 to 180	
11	99·2	—	88	1 to 191	
Patient left hospital on June 15th cured.					

This case is one of a different class, the observations having been made in a pneumonic patient. It shows a curious increase in number of the white blood-corpuscles up to the crisis of the disease as they gradually increased from 1 to 369 to 1 to 166.

At the crisis of the case there occurred a very sudden decrease in their number, the table showing a fall from 1 to 166 to 1 to 571, and then a gradual rise during resolution until they reached 1 to 191.

This may be the usual course in pneumonia, but it is the *only case* of this disease which I have estimated, and while it confirms the opinions of Virchow and Nasse as to the increase of white blood-corpuscles in pneumonia it can be taken as proving nothing further.

Case 17.—Case of typhoid fever, with constipation. Observations commenced on first day of illness.

Date. March	Temp. a.m.	Temp. p.m.	Per cent. No. of R.C.	Relative No. of W.C.	
8	102·4°	103·2°	90	1 to 300	
10	100·2	102·8	96	1 to 436	
15	99	103	92	1 to 511	Average 1 to 420.
17	101·2	103·4	94	1 to 313	
24	98	100·4	76	1 to 542	

After a relapse temperature became normal on March 26th. On April 7th there was slight periostitis of tibia.

April					
9	98·6	99·2	92	1 to 287	
10	98·4	99·2	92	1 to 418	
10	5.30 p.m. abscess opened		98	1 to 490	Average 1 to 390.
11	98·4	98·4	92	1 to 383	
12	—	98·4	98	1 to 376	

Case 18.—A case of typhoid, accompanied by high fever, great delirium. Death on twenty-second day of illness. Post-mortem showed extensive and well-marked ulceration of intestine. Observations commenced on twenty-second day of disease.

Date. June	Temp. a.m.	Temp. p.m.	Per cent. No. of R.C.	Relative No. of W.C	
3	99	104	100	1 to 652	
4	102·2	103·6	86	1 to 537	
5	103	104·6	86	1 to 537	
6	103·6	104·2	80	1 to 363	
7	101·6	104	96	1 to 480	Average 1 to 486.
8	102	104·2	98	1 to 445	
9	103·2	105·2	88	1 to 366	
11	102	104·2	82	1 to 512	

Cases 17 and 18 are two ordinary cases of typhoid fever, both of which show a very decided decrease in the relative numbers of the white blood-corpuscles, in Case 17 the proportion being 1 to 420, and in Case 18 1, to 486.

But again, these results depend on two cases only, and as the patients were in each case delirious, some difficulty was experienced in obtaining the blood necessary for the estimations, so that an error may easily have occurred. This may account for the fact that the above results differ from those given by Virchow, who states, "that the white corpuscles are increased in the typhoid state."

Case 19.—Case of acute rheumatism. Slight effusion in left wrist and left ankle.

Date.	Temp.		Per cent. No.	Relative No.	
March	a.m.	p.m.	of R.C.	of W.C.	
24	101·8°	102°	88	1 to 366	
26	98·8	98·4	94	1 to 313	
27	98·2	98·2	84	1 to 420	
28	98·2	99	88	1 to 314	Average 1 to 345.
29	98·6	98·4	94	1 to 427	
31	99·8	100·2	84	1 to 233	
April					
1	99·8	100·4	90	1 to 225	
2	100·2	101	72	1 to 156	
3	99	101·2	96	1 to 252	
4	99·2	101·6	76	1 to 316	Average 1 to 244.
5	99·8	100·4	86	1 to 252	
8	99	—	96	1 to 266	

This last case is one of rheumatic fever, in which there was slight effusion into the left wrist and ankle.

In the first half of this case there is no increase at all in the number of the white blood-corpuscles, which for six estimations on separate days gives an average of 1 white to 345 red corpuscles, while in the latter half of the case there is a slight increase in their number, the average of six observations being 1 white to 244 red corpuscles.

It is only included in this paper because to a certain extent it corroborates the view suggested by Cases 14 and

15 that serous inflammations do not cause an appreciable increase in the relative numbers of white blood-corpuscles.

The preceding are the cases in which I have made estimations of the number of white corpuscles; they include, as we have seen, the following:

Case 1. Iliac abscess.
„ 2. Pelvic cellulitis and probably abscess.
„ 3. Suppurating white leg.
„ 4. Suppurating tonsillitis.
„ 5, 6. White swelling treated by the actual cautery.
„ 7, 8, 9, 10. Empyema.
„ 11, 12, 13. Phthisis.
„ 14, 15. Serous pleurisy.
„ 16. Lobar pneumonia.
„ 17, 18. Typhoid fever.
„ 19. Acute rheumatism.

In Cases 1 to 4 (abscesses), and 7 to 10 (empyemas), where there was suppuration with pent-up pus, we have in each individual case a marked increase in the number of the white blood-corpuscles so long as this tension remained, but as soon as the pus was evacuated and free drainage established, the number of white corpuscles returned practically to normal.

In Cases 11 and 12 (phthisis) we have suppuration with fairly efficient, but not complete, drainage, and there is corresponding slight increase in the white blood-corpuscles such as we have seen in Case 1 while the abscess cavity was closing by granulation.

However, if we turn to Case 13 (phthisis and psoas abscess), we at once see a much larger relative increase, due probably to the inefficient drainage, for we have seen this same increase in Cases 3 and 7, which were acknowledged to be badly drained.

If we now look at the cases of inflammation of serous membranes accompanied by serous or sero-fibrinous exudation we find very different results.

Cases 14 and 15, which are serous pleurisies, and Case

19, which is a single case of acute rheumatism, show that not only is there no increase in the white corpuscles, but in the pleurisy cases there is even actual decrease during convalescence.

Case 16 (pneumonia) stands by itself, and I can offer no explanation concerning it, as the patient left the hospital and was lost sight of while he still had a large increase of white blood-corpuscles.

In the typhoid cases (Nos. 17 and 18) there was, as we have seen, a large decrease in the numbers of the white blood-corpuscles; this may be usual, but I have explained previously one very possible source of error in these cases.

Cases 5 and 6 are recorded, not to show that there is any increase in the number of the white corpuscles in cases of white swelling, but to show the effect of severe local irritation, and in both we see a decided increase, while the acute inflammation lasted, but this was lost as soon as free suppuration was established.

The above observations confirm the opinions of the continental observers quoted at the commencement of this paper as far as pneumonia and phthisis are concerned, and especially the single observation by Malassez on the decrease in the number of the white corpuscles when tension is removed.

On looking at these results, I think that we are justified in drawing the following conclusions:

1. That white corpuscles are increased in number in suppurative inflammations, especially when accompanied by tension.

2. That they are slightly increased in parenchymatous inflammations.

3. That they are not increased in inflammations accompanied by serous or sero-fibrinous exudations.

Concerning the pathology of the above increase, I do not propose to offer any details, but I would suggest that it may be due to absorption of leucocytes from the inflamed area in the neighbourhood of the abscess.

It has also occurred to me that the increase noticed in

the number of white corpuscles in the case of an empyema might be of diagnostic value if it proves on further observation to be constantly present.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' New Series, vol. ii, p. 37.)

A COMMUNICATION

ON THE

REMOVAL OF A GROWTH FROM THE BRACHIAL PLEXUS,

AFFECTING THE ROOTS OF THE EIGHTH CERVICAL AND FIRST DORSAL NERVES AT THEIR EMERGENCE FROM THE INTERVERTEBRAL FORAMINA.

BY

EDWARD BELLAMY, F.R.C.S.

Received September 9th, 1885—Read January 12th, 1886.

Tumours associated with the large nerve-trunks of the cervical and brachial plexuses are comparatively rare, and a successful removal, with complete restoration of functions, possibly still more so.

The following case has therefore been considered worthy of record—not only surgically, but as having several points of physiological interest.

On Nov. 11th, 1884, I saw, in consultation with my colleague, Dr. Mitchell Bruce, a lady, fifty-four years of age, of considerable embonpoint, who suffered from a growth in the root of the neck.

The swelling occupied the right side of the neck, and

sprang from the base of the subclavian triangle, and was evidently very deeply seated. It was of doubtful mobility, and caused the patient very great pain when manipulated, however gently. I learned that, for several years past, the patient had complained of tingling and pain shooting down the arm and rendering it useless, whilst her health was becoming seriously affected.

About two years ago she noticed the present growth, appearing just above the centre of the right clavicle, and becoming especially evident when the shoulder was depressed. It steadily but very slowly increased in size. All the muscles supplied by the ulnar nerve appeared to be affected, both in the forearm and hand, whilst the areas supplied by the cutaneous branches of this nerve, and indeed of the entire inner cord of the brachial plexus, were excessively sensitive.

There was, moreover, extreme sensibility on the lateral aspect of the thorax; apparently corresponding with the intercosto-humeral nerve.

The integument supplied by the cutaneous branches was œdematous and shiny, and the veins somewhat congested.

The exact diagnosis of the nature of the case was manifestly difficult,—whether there was a tumour of, or in some portion of, the brachial plexus; whether the symptoms were the result of pressure from a growth dissociated with the nerves; or whether the swelling was possibly due to a consolidated aneurismal sac. The symptoms, however, pointed to a growth involving the root of the ulnar nerve.

It was deemed advisable to discover the real nature of the growth, and if possible to remove it.

Operation.—On November 17th, assisted by Mr. Stanley Boyd, after drawing down the integuments, I made a linear incision along the clavicle, as in ligature of the subclavian, but finding I had no room, I converted this into a ⊥ incision, the vertical portion of which ran up along the posterior border of the sterno-mastoid. Some few superficial veins were cut and tied. Arrived at the

omohyoid, I hooked it up out of the way, and proceeded to define the growth with my fingers. Some portions of the plexus came into view, clearly placed over it, and apparently somewhat "frayed" out. On these nerves being hooked aside an encapsuled growth, smooth on the surface, in shape like a chestnut, and having one pole adherent to the scalene muscles was exposed. This pedicle was cut through with scissors, when a small nerve was divided. The under surface of the growth partly rested on the first rib and pleura, and partly encroached upon the combined cords of the last cervical and first dorsal nerves, at their emergence from between the scalene.

The growth was then readily peeled off a nerve of no great size, perhaps the suprascapular, perhaps the anterior thoracic, but certainly not one of the great cords, whilst the subclavian artery lay at its base inside. This vessel was carefully hooked on one side. The chief attachment of the growth was towards the scalene muscles. Owing to the fatness of the neck the tumour was very deep, but the entire operation occupied but a very short time. A drainage-tube was put in, the edges of the wound approximated, a pad of salicylic wool placed over all, and the arm brought across the chest. (The wound was completely healed on the seventh day and the temperature never rose above 99° F.). On the day after the operation the patient stated that, although in some pain, it was of a very different character to that she had experienced before the operation was performed.

Progress of the Case.—Shortly after the operation some symptoms of paralysis of the muscles of the arm and shoulder came on. The patient could not grasp with the fingers nor rotate the elbow-joint, and she was unable to lift the arm from the side. As this condition became more marked, it was decided to apply the constant current, and this was obtained by the ordinary Leclanché battery, thirty cells' strength. Galvanism was at first productive of little or no good. But under the care of Dr. Risk, of Harrow, by great attention to the application of the current, the

functions of the arm began slowly to return. At the present date (July, 1885) the patient has complete control over the arm, forearm, and hand, perfect sensibility, and complete freedom from pain.

Nature of the Growth.—The following is the account of the examination of the tumour by my colleague, Mr. Stanley Boyd:

"The tumour has the shape of a flattened sphere, one inch in its longest diameter, well encapsuled, smooth on the surface, having one pole adherent to the scalene muscles, and it was at this point on cutting through its pedicle with scissors, that a nerve seemed to be divided. A few nerve-fibres were found spread out on the superior aspect of the growth, but none penetrated the capsule, and there was no trace of nerve-structure in it on section.

"The section had a greyish or yellowish, more or less translucent appearance, the soft tissue being intersected by distinct bands of fibrous tissue, stronger and better marked inferiorly than elsewhere; superiorly almost all the tissue was soft and translucent. A scraping examined in water showed the tumour to consist largely of round cells, about as large on the average as white corpuscles. The nucleus was rarely seen, and most of the cells contained a few fat cells; granular oval cells were common, perhaps representing spindle-cells with processes torn off. Small shreds consisted chiefly of closely-packed cells, arranged in groups separated by bands of fibrous tissue or of spindle-cells.

"A section stained and examined under the microscope shows the growth to consist of closely-packed cells of the above form, the spindle-cells forming broad bands between groups of round or of spindle-cells cut transversely. For the most part the substance between the nuclei is in small amount and obscurely fibrillated, thus producing the whitish bands visible to the naked eye. In this denser tissue small irregular spaces, formed probably by mucous degeneration and containing clear coagula, are sometimes seen. The vessels are tolerably numerous, and of con-

siderable size; their walls are formed by the tissue of the growth. No nerve-fibres were detected.

"The tumour is therefore of the common connective-tissue type, showing but a slight departure from the embryonic condition. Had it infiltrated surrounding tissues it would unhesitatingly be classed as a sarcoma, but if this is its anatomical position the presence of a capsule and the ease with which the growth shelled out, afford ground for believing that it will not recur."

Since the above was written I have received the following note from the patient's medical attendant in the country:

"On Feb. 20th, this year (1885), she had an attack of hemiplegia (right) owing to an embolic plugging of her left cerebral artery.

"The result of this was a return to nearly a similar condition of the arm as when she first came under my care. By the end of July, however, with the aid of occasional applications of the electric current, &c., she was able to sew, cut up her food, and write with a pen very fairly well, besides having considerable muscular power. From that date brain-irritation began to show itself, and finally culminated in an attack of acute mania. She died about the end of October from the effects of another grave brain-lesion, I believe in all probability profuse hæmorrhage into the medulla oblongata.

"But for these important complications I believe the case would have been ultimately successful."

NOTE BY J. MITCHELL BRUCE, M.A., M.D., F.R.C.P.LOND.

The leading feature of this case was *pain*, either of the nature of "a sort of soreness," increased by movement, so that the forearm had to be supported by the other hand, or of "a sudden, jerking, neuralgic" character, confined to the ulnar area of the hand. This pain was

accompanied by violent aching of the whole hand and forearm when muscular movements were attempted. There was also some itching of the ulnar border of the hand. No muscles were ascertained to be affected beyond those supplied by the ulnar nerve; but both the degree and the progress of the paralysis were difficult or impossible to determine exactly, on account of the severe pain which prevented or limited voluntary movements. No affection of the pupil was ever observed.

There can be no reasonable doubt but that the patient's death was entirely unconnected with the tumour or with the operation, and that it was due either to embolism or to cerebral hæmorrhage. The patient had been for an indefinite period the subject of aortic obstruction, and the cardiac action was irregular during the whole of the time she was under our observation. She had several attacks of incomplete paralysis, with disturbances of consciousness, before the fatal seizure; in one of them there was marked aphasia. (*May*, 1886.)

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' New Series, vol. ii, p. 41.)

STATISTICS OF MORTALITY IN THE MEDICAL PROFESSION.

BY

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Received October 15th, 1885—Read January 26th, 1886.

The mortality of the medical profession is a matter in which we are doubly concerned, having in it a personal as well as a scientific interest, so that no apology is required for bringing the subject before this Society; although I fear that some of the figures which I shall have to produce are scarcely calculated to give us much satisfaction.

When the census was taken in April, 1881, there were present in England and Wales 15,091 medical men, with the age-distribution shown in the first line of figures in Table 1. When the census was over, the death-registers for the whole of England and Wales were carefully gone through for three entire years, namely, 1880, 1881, and 1882, and the deaths of all males of fifteen years of age and upwards were abstracted with distinctions of age and occupation. In the second line of Table 1 are seen the results of this process so far as concerns medical men. On these data it is of course an easy matter to calculate the mean annual death-rate in each age-period, and the rates thus obtained are given in the last line of the table. The deaths thus recorded at the several age-periods give a total rate of 25·53 deaths annually to 1000 medical men of all ages

over twenty years. The mean annual death-rate of medical men between twenty and twenty-five years of age was 7·40 per 1000; between twenty-five and forty-five was 11·57 per 1000; between forty-five and sixty-five was 28·03 per 1000, and over that age was 102·85 per 1000.

TABLE 1.

Medical men.	Age-periods.				
	20–	25–	45–	65–	All ages.
Enumerated in 1881.	810	8300	4435	1546	15091
Died in 1880-1-2	18	288	373	477	1156
Mean annual mortality per 1000	7·40	11·57	28·03	102·85	25·53

Now, the first question which naturally suggests itself is whether this death-rate is a high or a low rate, and this question again divides itself into two; firstly, Is this rate, which is based on the records of the three years 1880-1-2, high in comparison with the mortality rate of medical men in former times? and secondly, Is it a high rate as compared with the death-rate of men engaged in other occupations?

To the first of these two questions the data collected in 1861 and in 1871 by Dr. Farr, my distinguished predecessor at the General Register Office, enable us to frame an answer. The annual death-rates deduced from those data are given in Table 2, and it will be seen in the last column of that table that while the annual death-rate of medical men over twenty years of age was 23·63 in 1860-1, it was 24·99 in 1871, and finally rose to 25·53 in 1880-1. These death-rates, let it be observed, are corrected for any difference in age-distribution at the successive periods; they are the death-rate for 1000 medical men, having the same age-distribution as existed in 1881.

TABLE 2. *Mean Annual Death-rates, per* 1000, *of Medical Men at Successive Dates.*

Date.	Age-periods.				Per 1000 with age-distribution as in 1881.
	20–	25–	45–	65–	
1860-1	5·86	12·78	23·47	91·69	23·63
1871	11·17	13·85	24·56	93·30	24·99
1880-1-2	7·40	11·57	28·03	102·85	25·53

It thus appears that there was a progressive increase in the mortality of the medical profession in the interval between 1861 and 1881, and our first question is therefore answered. The death-rate of the profession in 1880-1-2, was a high rate as compared with that of earlier periods.

It will be noted, however, on looking at the figures in Table 2, which gives the death-rates at each period of life, that the rise of the death-rate in 1880-1-2 as compared with 1871 was entirely due to an increase of mortality at the later periods of life, and that the mortality *fell* among medical men under forty-five years of age, while it *rose* in those of more advanced age. The same is also practically true when the 1880-1-2 rates are compared with those of 1860-1; for, though the death-rates of those who were between twenty and twenty-five years of age was lower in 1860-1 than in 1880-1-2, yet the proportionate number of medical men of that age is so small, only 54 in 1000, that the changes in the death-rate at that age scarcely affect the total result, that is to say are barely appreciable in the death-rate per 1000 at all ages over twenty.

It appears then that the mortality of medical men has increased at the later ages, namely, after forty-five years, while it has diminished at the earlier ages. The increase at the later ages has, however, been greater than the diminution at the earlier ages, and consequently the total result has been an increased death-rate.

This increase of mortality at the later ages, and this decrease of mortality at the earlier ages, was not peculiar to the medical profession. A similar increase and decrease occurred in the mortality of many, and indeed of most, other professions and industries, and was in fact the most noticeable phenomenon presented by the mortality of males generally in this country in the last decennium. What was exceptional in the medical profession as compared with most other occupations was this, that in that profession the increased mortality at the later ages was greater than the diminution at the earlier ages, whereas in most other occupations, and among the male population generally, independently of occupation, the contrary was the case, and the lives saved at the earlier ages were in excess of the lives lost at the later ages.

To the question, which cannot but present itself, why it is that there has been this strange increase of mortality among the male population of this country at the later age-periods coincidently with a decrease of mortality in the earlier stages of life, only a conjectural answer can be given. Two causes can be pointed out that must almost certainly have contributed to produce this result, and that not impossibly may account for the whole of it. Firstly, there is the increased wear and tear of adult life, brought about by the growth of population and by the keener struggle and competition which this growth necessarily brings with it. Secondly, the very efforts that have been made with such marked success to improve the sanitary condition of the country, while they have enormously reduced the mortality of the young, may very probably have tended to increase the mortality at the later ages; for a large number of comparatively weak lives, which in pre-sanitary times would have perished in infancy or childhood, have been preserved, and by their survival must have diminished the average vitality of the population of more advanced ages. The question, however, of the causes of the strange changes that have occurred in the male death-rates, though it is one which it was im-

possible to pass over altogether in silence, in reality lies almost outside the present inquiry; for the changes affect the whole male population, whereas the present inquiry relates specially to the medical profession.

Let us pass on therefore to the second part of our first question, Was the mortality of medical men in 1880-1-2 not only high when compared with their mortality in former times, but also high as compared with the mortality of men engaged in other occupations?

The answer to this question will be found in Table 3, which gives in the first column of figures the annual death-rates in 1880-1-2 for a number of different professions and trades, the death-rate being in each case calculated for 1000 males over twenty years of age, with an age-distribution similar to that of the medical profession.

TABLE 3. *Mean Annual Death-rates of Males in different Occupations corrected for Differences in Age-Distribution.* 1880-1-2.

Profession, Trade, or Industry.	Annual death-rate per 1000.		Profession, Trade, or Industry.	Annual death-rate per 1000.	
	Males 20 years of age and upwards.	Males 25 to 65 years of age.		Males 20 years of age and upwards.	Males 25 to 65 years of age.
All occupations .	22·83	15·42	Watch, Clock, Philosophical Instrument		
Medical Profession	25·53	17·30	Maker, Jeweller .	21·20	14·36
Clerical „	15·93	8·57	Printer . .	23·75	16·51
Legal „	20·23	12·97	Bookbinder . .	25·36	18·00
Schoolmaster .	19·90	11·09	Earthenware Manu-		
Clerk (Commercial and Law) .	21·10	15·61	facturer . .	35·98	26·83
Commercial Traveller	20·06	14·61	Cotton Manufacturer	27·19	16·76
Farmer . .	17·49	9·78	Woollen, Worsted		
Agricultural Labourer	18·28	10·80	Manufacturer .	26·47	15·91
Gardener . .	15·08	9·24	Mason, Bricklayer,		
Innkeeper, Publican .	29·02	23·47	Builder . .	22·29	14·94
Brewer . .	29·23	20·99	Carpenter, Joiner .	19·30	12·64
Chemist . .	22·52	15·66	Painter, Plumber, Gla-		
Shopkeeper .	19·43	13·52	zier . . .	25·95	18·53
Butcher . .	25·89	18·05	Cutler . . .	28·52	20·18
Baker . .	21·87	14·77	Blacksmith . .	23·14	14·99
Tailor . .	22·45	16·21	Quarryman . .	26·42	17·29
Shoemaker . .	20·66	14·20	Coalminer . .	23·97	13·72

A glance at this table at once shows that the death-rate in the medical profession is far in excess of the death-rate in any one of the other professions which can be most fitly put into comparison with it. Thus the death-rate in the clerical profession is only 15·93, in the legal profession is 20·23, in the scholastic profession is 19·90, while in the medical profession, as before stated, it is no less than 25·53. Nor is the medical death-rate higher only than the rate in the other learned professions; it is higher than the rates in most trades and industries, higher, for instance, than those of chemists, shopkeepers, bakers, tailors, shoemakers, blacksmiths, carpenters, coalminers, and of many other groups in the table, and is only itself exceeded by the rates in certain trades and occupations that are notoriously unhealthy.

There is, it is true, some little unfairness in the comparison of the death-rates in the medical and other learned professions with the death-rates in other occupations. A medical man, or a clergyman, when he has once adopted his profession, remains in it, as a rule, for the rest of his life; and at whatever age death may overtake him it will almost certainly find him still calling himself a member of his profession, though he may have abandoned all practical exercise of it. The death of a medical man, therefore, or of a clergyman, will almost certainly be registered as such, however old the deceased may have been. But with most other occupations such is not the case; a man engaged in one of them, when he becomes incapacitated for active work by disease or old age, gives up the occupation, and with it very often gives up the name. The death of such a man would be registered without specification of the occupation which he had followed in his active days. On this account, as also for some other reasons which it is unnecessary here to state, it is better, when comparing the death-rates in different occupations, to limit the comparison to males in the great working period of life, namely, in the four decennia that lie between the completion of the twenty-fifth and the

sixty-fifth years of life. A column has consequently been added to the table in which are given the death-rates in each profession and trade per 1000 males in this period of life, the age-distribution being as before assimilated to that which existed in 1881 in the medical profession. That is to say in each case the death-rate is that of 1000 males, of whom 652 were in the first half (25—45) and 348 were in the second half (45—65) of the whole age-period of forty years. The relative mortalities in the several occupations, as shown in this column, differ in some instances very considerably from the relative mortalities for the more extended age-period, as given in the earlier column. But the results, so far as our present purpose is concerned, remain unaltered; the medical death-rate, as before, is far in excess of the rates in the other learned professions, and also of the rates in most trades and industries. Do then what we may, we cannot avoid the unpleasant conclusion that the death-rate of medical men is excessively high, and this whether we compare it with the death-rate in the same profession at earlier dates, or with the death-rates of men engaged in other professions and in most trades and industries.

There are, of course, within the compass of the medical profession sub-groups of practitioners that differ very widely from each other in the social and other conditions under which they live. The life and habits, for instance, of a London physician or surgeon differ enormously from the life and habits of a practitioner in some out-of-the-way rural district, and these again from the life and habits of a surgeon in the army or in the navy; and were it possible to separate these sub-groups accurately from each other and to calculate their death-rates severally, it would assuredly be found that such rates would present wide differences of amount; and in this connection it may be pleasant to the Fellows of the Royal Medical and Chirurgical Society to be reminded, that, some thirty or more years ago, the records of the Society from its establishment in 1805 to the beginning of 1851 were inves-

tigated by a distinguished actuary, the late Mr. F. G. P. Neison,[1] and that it appeared from his calculations, that the mortality in the Society approximated very closely to that of the male population of England and Wales, or indeed was fractionally below it, the actual number of deaths among the Fellows having been 96, whereas it would have been 97·1 had their rate of mortality been equal to that of all males of corresponding ages in England and Wales. In strong contrast with this were the death-rates among the medical men in the army. Here the mortality, as calculated by Mr. Neison, was very greatly above that of the whole male population, the general result of his inquiries being, that out of equal numbers living and having the same age-distribution, there were for the general male population 100 deaths, for the army medical men 162 deaths, and for the Fellows of the Royal Medical and Chirurgical Society 99 deaths.

The existence of such differences of mortality within the profession itself is of course a matter of considerable interest and importance. The data, however, on which the present inquiry is based do not permit of any breaking up of the medical profession into sub-groups, and consequently in this paper the profession can only be dealt with in the aggregate, and treated as a homogeneous whole.

Having now seen that the mortality in the medical profession is extremely high, let us proceed to consider what are the causes to which this is attributable, or rather what are the diseases under which the excess of mortality occurs.

Let me first state what are the data by means of which I propose to examine this question. The local registrars of deaths throughout the country have directions, whenever they register the death of a medical man, to send up a full copy of the entry to the Medical Register Office, in order that the Medical Register may be duly corrected by the erasure of the deceased man's name. As a check on

[1] Cf. 'Contributions to Vital Statistics' (1857), p. 102.

the local registrars, who are paid half-a-crown for each such entry when transmitted, copies of such entries are also forwarded to the General Register Office at Somerset House, and I have availed myself of the accumulations of these copies of entries for my present purpose, and have been thus enabled to present to the Society a table which is, I think, unique, no similar table existing either for the medical or any other profession or industry.

TABLE 4. *Registered Causes of Death, with Ages, of* 3865 *Medical Men.*

Causes of death.	Age-periods.								All ages.
	20–	25–	35–	45–	55–	65–	75–	85 and upwards.	
Smallpox	...	1	...	1	...	...	...	...	2
Scarlet Fever	...	6	2	...	1	...	...	...	9
Typhus	4	5	1	1	...	1	...	...	12
Diphtheria	3	3	2	...	...	1	...	...	9
Simple continued Fever	...	2	1	...	1	1	...	...	5
Enteric Fever	1	18	8	5	9	5	1	...	47
Cholera	...	...	...	1	1	...	...	...	2
Diarrhœa	...	4	1	2	3	7	8	4	29
Malarial Fever	...	2	1	4	...	...	...	...	7
Erysipelas	...	3	6	8	2	6	1	...	26
Pyæmia, Septicæmia	...	2	2	3	2	2	...	...	11
Venereal Affections	...	...	1	1	...	1	...	...	3
Alcoholism	...	8	13	4	2	...	...	...	27
Rheumatic Fever and Rheumatic Affections of Heart	1	5	9	8	6	1	...	...	30
Rheumatism	...	1	...	1	2	3	1	...	8
Gout	...	1	1	9	13	11	9	...	44
Cancer	...	...	11	15	42	55	10	...	133
Phthisis	13	88	83	37	31	10	1	...	263
Diabetes Mellitus	...	...	3	5	15	19	1	...	43
Inflammation of Brain	...	2	3	3	2	3	...	1	14
Softening of Brain	1	2	6	13	25	35	19	1	102
Apoplexy	...	7	14	32	52	83	41	8	237
Paralysis	...	1	6	12	34	45	31	2	131
Paraplegia, Disease of Cord	...	5	4	10	12	16	2	...	49
Epilepsy	...	8	9	4	6	8	2	...	37
Insanity, General Paralysis of Insane	...	6	20	11	8	7	3	...	55
Other or undefined Diseases of Nervous System	...	6	13	8	17	16	5	1	66
Endocarditis, Valvular Disease, Pericarditis	1	9	8	13	22	32	11	1	97
Hypertrophy of Heart	...	1	...	...	3	6	1	...	11
Angina Pectoris	...	1	1	5	9	13	5	...	34

Causes of death.	Age-periods.								All ages.
	20–	25–	35–	45–	55–	65–	75–	85 and upwards.	
Aneurysm	...	4	11	7	6	4	...	...	32
Embolism	...	...	...	3	3	2	1	...	9
Other or undefined Diseases of Heart and Circulatory System	...	18	22	49	123	143	78	11	444
Bronchitis	...	6	14	11	32	48	54	14	179
Pneumonia	2	24	46	29	34	36	9	1	181
Pleurisy	...	3	3	1	5	4	1	1	18
Asthma, Emphysema	...	3	2	5	7	10	4	...	31
Laryngitis	...	1	4	1	1	2	1	1	11
Other and undefined Diseases of Respiratory System	...	9	9	7	13	13	14	5	70
Ascites	...	...	...	3	...	2	1	...	6
Gall-stones	...	...	...	...	6	2	...	1	9
Cirrhosis of Liver	...	3	20	31	27	9	...	...	90
Other or undefined Diseases of Liver	1	13	31	32	31	35	14	2	159
Diseases of Stomach	...	5	4	11	8	14	6	...	48
Hæmatemesis, Melæna	...	1	5	4	1	5	...	...	16
Enteritis	...	...	2	1	2	1	1	2	9
Ulceration of Intestine	...	2	3	4	2	5	...	...	16
Ileus, Obstruction, Stricture, Strangulation of Intestine	...	...	...	3	4	13	9	2	31
Hernia	...	...	...	...	...	1	1	...	2
Fistula	...	...	...	1	2	...	...	...	3
Peritonitis	...	2	2	1	1	3	...	...	9
Other or undefined Diseases of Digestive System	...	...	2	3	4	3	3	...	15
Nephritis	...	2	2	1	2	1	1	...	9
Bright's Disease	1	7	30	33	35	34	12	...	152
Calculus	...	...	2	3	6	1	1	...	13
Hæmaturia	...	1	1	...	...	3	1	...	6
Suppression of Urine, Uræmia	...	3	3	1	3	4	3	2	19
Diseases of Bladder and Prostate	...	...	2	1	11	42	38	2	96
Other or undefined Diseases of Urinary System	...	6	9	7	8	9	3	2	44
Caries and other Affections of Bones and Joints	...	3	2	3	5	3	...	...	16
Carbuncle	...	...	...	2	1	6	...	...	9
Old age	...	...	...	...	2	52	132	68	254
Accident	1	18	33	24	16	20	6	2	120
Suicide	2	14	11	16	5	4	3	...	55
Other or undefined causes	3	14	16	23	35	25	22	3	141
Total	34	359	520	537	761	946	571	137	3865
Age-periods	20–	25–	35–	45–	55–	65–	75–	85 and upwards.	All ages.

This table gives the registered causes of death in combination with age for no less than 3865 medical men, who died at some time within the ten years 1873-82, and the certificates of whose deaths have been preserved.

Seeing how large a number 3865 is, and seeing that these deaths came from all parts of the country indifferently, and were moreover spread over a period of ten years, we may assume with much confidence that they are a perfectly fair sample, representing with close accuracy the bulk of the mortality of medical men, when distributed by causes and by ages. Now, it has already been shown that the mean annual mortality of medical men in 1880-1-2 was 25·53 per 1000, or—as it will be convenient to avoid the use of decimals—25,535 per million. We can therefore divide out the 25,535 deaths, which occur annually among a million living medical men, in the proportions given us by the 3865 deaths of which we have the causes; and by so doing we shall of course have the annual death-rate per million from each separate cause so dealt with.

The results are given in the first figure column of Table 5; while the second column of figures gives for comparison the corresponding rate for all males in England and Wales irrespectively of occupation, due correction having been made for difference of age-distribution. That is to say, the rates for all males are the annual rates for a million males, with the same age-distribution as existed in the medical profession in 1881. That distribution was as follows:

20 and under 25 years	= 53,674
25 „ 45 „	= 549,997
45 „ 65 „	= 293,884
65 and upwards	= 102,445
Total . .	1,000,000

TABLE 5.

Causes of death.	Annual deaths per million living males over 20 years of age.		Causes of death.	Annual deaths per million living males over 20 years of age.	
	Medical men.	General population.		Medical men.	General population.
Smallpox	13	78	Diseases of Circulatory System	4142	2934
Scarlet Fever	59	16	Diseases of Respiratory System	3237	4408
Typhus	79	38	Liver Diseases	1744	744
Diphtheria	59	14	Other Diseases of Digestive System	973	632
Simple or ill-defined Continued Fever	33	40	Calculus	86	30
Enteric Fever	311	238	Diseases of Bladder and Prostate	634	287
Diarrhœa, Cholera	205	274	Other Diseases of Urinary System	1520	665
Malarial Fever	46	11	Hernia	13	88
Erysipelas	172	136	Accident	793	1105
Alcoholism	178	130	Suicide	363	238
Gout	291	78	All other causes	2869	2124
Rheumatic affections	251	215			
Malignant diseases	879	790			
Phthisis	1738	3145			
Diabetes	284	108			
Diseases of Nervous System	4565	4268	Total from all causes	25,535	22,829

It will at once be seen that the figures in the two columns differ very widely; and the general result of the comparison is to show that, with very few exceptions, the mortality of medical men is higher under every heading than the mortality of males generally, and that under some of the headings the medical mortality is twice or thrice, or even more times, greater than the average.

There are altogether in the table twenty-seven headings, and in only seven out of these is the medical death-rate lower than that of males generally. Moreover, of these seven headings under which the advantage is on the side of the medical men, there are but three of any numerical importance, viz. phthisis, diseases of the respiratory organs, and accident. Again, as regards the last-mentioned of these, namely, accident, although the mortality of medical men is very considerably below the average, this is only because the average is raised by the inclusion

in the general population of men employed in a small number of highly dangerous occupations, and, when these exceptionally dangerous industries are left out of the account, the death-rate of medical men from accident is, as will be shown later on, a high one. Thus there remain only two headings in the table, namely, phthisis and diseases of the respiratory system, under which the medical mortality is in any important degree lower than the average. The medical mortality from phthisis is 45 per cent., and from diseases of the organs of respiration 27 per cent., below that of the general male population. The advantage thus enjoyed by medical men is in all probability due rather to their social than to their professional position; phthisis and lung affections being diseases which are especially destructive among the classes that suffer from destitution; and the medical profession being of course, as compared with the general male population, a class in easy circumstances. Something also may fairly be put to the credit of the knowledge of the healing art which medical men who fall ill have at their command; and it is to this latter advantage, combined with the absence from their occupation of the necessity for any severe muscular strain or exertion, that the much smaller mortality of medical men, as compared with the general population, from hernia, is to be ascribed. The figure under this heading is for males generally 88, but for medical men only 13. To medical knowledge must also be attributed the fact that while the mortality of the general male population from smallpox is 73 per million, the mortality of medical men from that disease is only 13 per million. Medical men are not likely to be led astray in their own persons by the statements of anti-vaccinationists, and consequently, though they are of course much more exposed to the chance of infection, their mortality from smallpox is scarcely more than one sixth of the average; and this fact is the more striking, inasmuch as the reverse is the case with all those other infectious diseases against which no similar prophylactic

remedy is known, such as scarlet fever, diphtheria, typhus, enteric fever, and erysipelas. Under all these headings the mortality of medical men, as might be anticipated, is in considerable excess of the average. The slightly lower figure for medical men under the heading "Simple or ill-defined Forms of Continued Fever" is probably due to more than average accuracy of diagnosis, and more than average carefulness in statement of cause in the case of deceased medical men, who will scarcely ever have died without the presence of a brother practitioner.

The more than average mortality of medical men from remittent and intermittent fevers is attributable with much probability to the foreign element in the profession, that is to say, to the fact that a considerable number of army, navy, and other medical practitioners return to England from India and the colonies with diseases contracted in those parts.

Possibly the same explanation may account in some degree for the excessively high mortality of medical men from cirrhosis and other diseases of the liver, a mortality which is considerably more than twice as high as that of the general male population; but, seeing how great also is the excess of mortality in the profession under such headings as gout, alcoholism, and calculus, not to speak of diseases of other digestive organs than the liver, it becomes difficult to resist the conclusion that the main part of the enormous mortality from hepatic diseases is due, despite of the indignant protest of Professor Casper[1] to the contrary, to the neglect on the part of medical men, as a body, of those wise rules of diet which they lay down for the guidance of their patients.[2]

[1] 'Annales d'Hygiène Publique,' xi, 1834, p. 384.

[2] That doctors are prone to neglect in their own persons the rules of abstemiousness which they lay down for others is a charge of great antiquity, as is shown by the following fragment of Philemon:

τεκμήριον δε, τοὺς ἰατροὺς διὸ ἐγὼ
ὑπὲρ ἐγκρατείας τοῖς νοσοῦσι εὖ σφόδρα
πάντας λαλοῦντας, εἰτ' ἐὰν πταίσωσί τι,
ποιοῦντας ἀυτούς πανθ' ὅσ' ὀυκ ἐων τότε
ἰτέρους.

Scarcely smaller than the excess under the heading Liver Diseases is the excess of mortality in the profession from diseases of the urinary system. The liver-disease excess above the average for the general male population is 134 per cent.; the excess under the urinary headings is 128 per cent., or practically the same, for in calculations such as these, small differences are of course without much value. On the other hand the excess of mortality from diseases of the organs of circulation is only 41 per cent., and from diseases of the nervous system only 7 per cent., above that of the general population. Another disease in the table under which there is a remarkable excess is diabetes, the medical mortality from this disease being 284 per million living, while that of the general male population is only 108. The numbers are small, and consequently too much importance must not be attached to them; but I may point out that Dr. Richardson, I do not know on what basis of observed facts, mentions[1] diabetes as a disease to which medical men are especially liable, and explains this liability by the excessive nervous fatigue incident to a medical practice; and that other writers speak[2] of diabetes as a disease more common among the well-to-do classes than among the comparatively poor, who of course form the great bulk of the general population with which the medical profession is contrasted in our table.

There remains one other group of diseases in the table, namely, cancer and other malignant tumours, which requires notice before passing on to the mortality from violence. The medical mortality from cancer or malignant disease is 879 per million living, while the figure for the general male population is only 790, a difference of about 11 per cent. This apparent difference is not more, however, than can be rationally explained by the fact that the diseases to which medical men succumb are almost certain to be more accurately diagnosed and more carefully stated in death-certificates than the fatal diseases of the general population. A cancerous or malignant

[1] 'Diseases of Modern Life,' p. 408. [2] 'Ziemssen's Cyclop.,' xvi, 863.

tumour which proves fatal to a medical man will almost certainly be diagnosed as such, and its nature stated by his brother practitioner in the death-certificate, whereas among the poorer classes it too often happens that the nature of the tumour is not made out, and the cause of death is simply given as "abdominal tumour" without further specification.

Let us now pass on from the mortality caused by disease to the mortality from violence, accidental or suicidal. The table shows an annual mortality from accident for medical men of 793 per million living, while the figure for the general male population is 1105. But, as was previously mentioned, the figure for the general population is unduly raised by the inclusion of men engaged in a small number of highly dangerous occupations, such as mining, quarrying, and sea-fishing; and, as is shown in Table 6, which gives the annual mortality of males between twenty-five and sixty-five years of age in various trades and industries, the medical accident-rate is in reality high, for of the twenty other occupations in the table there are but eight in which the rates are higher.

TABLE 6.—*Mean Annual Mortality from Accident per Million Males, from Twenty-five to Sixty-five Years of Age,*[1] *in different Occupations*, 1881-2-3.

Occupation.	Rate per million.	Occupation.	Rate per million.
Miners	2785	Commercial Travellers	557
Fishermen	2351	Butchers	541
Quarrymen	2290	Agricultural Labourers	511
Cabmen	1299	Farmers	464
Painters, Plumbers, and Glaziers	1129	Cotton Workers	464
Blacksmiths	758	Wool, Worsted Workers	418
Builders, Masons, and Bricklayers	696	Gardeners	371
Innkeepers, Publicans	696	Pottery Workers	371
Medical Men	644	Bakers	325
Carpenters, Joiners	588	Tailors	278
		Shoemakers	263

[1] The rates in this table are for males between twenty-five and sixty-five

To what forms of accident is this high mortality among medical men to be ascribed? To this question it is impossible to give a thoroughly satisfactory answer, owing to the inadequate manner in which the nature of a fatal accident is too often stated in a coroner's certificate. The following table gives, however, the registered causes of the 120 fatal cases of accident that occurred among the 3865 deaths tabulated on pages 225, 226.

Registered Causes of 120 *Deaths of Medical Men from Accident.*

Railway accident	7	Laudanum, morphia	18
Carriages or horses	17	Chloroform	6
Cut	2	Nitrous oxide (tooth extraction)	1
Fall from height	1	Chlorodyne	1
Fall downstairs	4	Chloral hydrate	9
Other falls	7	Prussic acid	9
Burn	2	Carbolic acid	2
Gas explosion	1	Poison (kind unstated)	2
Lightning	1	Fracture	11
Sunstroke	1	Kind of accident not stated	8
Gelatio	1		
Drowning	7	Total	120

It will be seen that a not inconsiderable proportion of the 120 deaths, namely 17, were caused by accidents with carriages or horses, a kind of accident to which medical men, especially in rural parts, are, of course, much more exposed than the average of men; very probably, moreover, many of the fatal fractures and injuries, of which the causes are not given, may have been due to similar kinds of accident. But the most notable feature in the table is the overwhelming amount of accidental death from poison. In no less than 49 out of the 120 accidental deaths, or in 40 per cent. of the whole, the death was caused by poison, and in the great bulk of these cases

years of age, and are based on the data given in the 'Supplement to the Registrar-General's 45th Annual Report;' whereas the rates in Table 5 are for all males over twenty years of age.

the poison was either some or other form of anodyne or prussic acid. It must be remembered that in all cases in which a person is found dead, without distinct evidence of the circumstances under which the death occurred, the death is considered to be accidental; but that some, at any rate, among these numerous deaths from poison were not accidental can scarcely, I think, be considered an improbable or uncharitable hypothesis.

It remains to consider the mortality from suicide. The mean annual death-rate among medical men from this cause is given in Table 5 as 363 per million, while the figure for the general male population of corresponding ages is only 238, thus showing an excess of 52 per cent. on the side of medical men. Moreover, if in place of dividing out the total medical death-rate to the separate headings by means of the 3865 deaths in Table 4, which are spread over ten years, 1873—1882, the calculation of the suicide rate be made directly (as it chances there are means for doing) upon the deaths in the six years 1878—1883, it is found that suicide is apparently on the increase in the profession, for by this fresh calculation the suicide-rate for medical men rises to 464 per million instead of 363. The rate in Table 5, being based on a longer period of years, is doubtlessly the more trustworthy of the two; but I have been induced to give also the rate for the later and shorter period, because I am able for this period only to draw a comparison between the medical and the clerical and legal professions. The mean annual death-rate from suicide in the six years 1878—1883 was 123 among clergymen, priests, and ministers; 354 among barristers and solicitors; and, as already stated, 464 among medical men; in each case per million living and with the age-distribution of medical men in 1881.

It must, of course, not be forgotten that in treating of the annual mortality in any single occupation from suicide, as also from several others of the causes in the table, we are dealing with a small number of actual deaths, and that under such circumstances too much weight must not be

given to slight differences or slight fluctuations. But while on this account it would be unwise to insist upon the figures now given being taken as representing the constant proportions of suicides in the three great professions, they can, I think, be accepted without hesitation as showing that this mode of death is far more common in the medical than in the other professions. The figures represent accurately the proportions for six years, and in all probability the proportions would not be found very different if we had the data for a much longer period.

As regards the methods of self-destruction selected by medical men the most notable point is their preferential choice of poison. Out of the 55 cases of suicide in Table 4 26, or 47 per cent., were brought about by poison, and in no less than 15 of these 26 the poison used was prussic acid. This is what might have been expected, for medical men have free access to poisons, are familiar with their effects, and know which are the most expeditious and cause the least suffering.

Such are the statistics, so far as I have been able to ascertain them, of the mortality in recent years among the members of our profession. The figures, as was said at the beginning of this paper, are not such as to give us unmixed satisfaction. The ancient belief, which for ages was accepted by the general public and was supported by the theses of learned writers, that the life of a medical man was as a rule longer and freer from disease than that of an ordinary individual, inasmuch as, when in health, he guided his steps by the laws of hygiene, and when in sickness had the advantage of the best advice, after scarcely surviving the ridicule of Voltaire, received its death-blow so soon as the pitiless test of statistical inquiry was applied to the subject. But though figures, such as those I have brought before the Society this evening, are utterly incompatible with that ancient optimistic view, it is at any rate not unsatisfactory to note, that my figures give on the whole a much less gloomy view of the condition of the

profession than those put forth by some previous inquirers. Thus, Escherich,[1] writing some thirty years ago, stated that 75 per cent. of medical practitioners die before they reach the age of fifty, and more than 90 per cent. before they have completed their sixtieth year. But the figures given in my 4th Table show that instead of 75 per cent. dying before the age of fifty only 37 per cent. die before the more advanced age of fifty-five; and that instead of 91 per cent. dying before the age of sixty only 57 per cent. are gone before the age of sixty-five. Professor Casper[2] gives figures which are somewhat less appalling than those of Dr. Escherich, but nevertheless are much less favorable than those given in this paper. Casper, writing in 1834 of medical men in Germany, states that only 24 per cent. of them reach the age of seventy, this being a smaller percentage than in any other liberal profession. My figures (Table 4) show that 42·8 per cent. reach the age of sixty-five, and 18·3 per cent. the age of seventy-five; and calculating from the most recent life-table[3] this would mean a survival of 30·7 per cent. at the end of the seventieth year of life instead of only 24 as in Casper's estimate. Again, the average duration of life of the 624 medical men who formed the basis of Casper's calculation was 56·4 years, while the average duration of life of the 3865 medical men in my table was 59·3 years.[4] It is not then forbidden us to hope that some future statistician, when another few decennia shall have passed away, may find that the figures of his date may present a like improvement upon those which I have

[1] Cf. 'Dict. Encycl. des Sc. Médicales,' 2nd Sect., Tome v, 576.

[2] 'Annales d'Hyg. Publ.,' xi, 1834, p. 375.

[3] Cf. 'Suppl. to 45th Ann. Rep. of the Registrar-General,' p. vi.

[4] Dr. Guy ('Journ. of Statist. Soc.,' ix, 346) gives figures of apparently a much more favorable character than any quoted in the text. But Dr. Guy's calculations as to the mean duration of medical life were based exclusively on the deaths recorded in the Annual Register; and these would, as a rule, be only the deaths of such medical men as had attained some eminence in their profession, who, of course, would on the whole be of more than average age.

given, and that the sting may by that time have vanished from the old proverb—Physician, heal thyself.

(For a report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' New Series, vol. ii, p. 45.)

ON THE TAPETUM LUCIDUM.

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No satisfactory account has yet been given of the use of the tapetum lucidum, nor has its disposition in different animals been accurately described.

The tapetum has generally been examined after the eye has been removed from its socket. It is then difficult to replace it in its exact natural position, and it has consequently been generally loosely described as irregularly placed at the back, or outer part of the back of the eye.

In order to ascertain the exact position of the tapetum, in its relation to surrounding parts, it should be examined *in situ*, before the eyeball is removed. For this purpose the upper part of the orbit may be taken away, leaving the eye in its natural position. The anterior part of the eye, including the iris, must also be removed, any colouring matter that may have escaped from the choroid must be washed out with a thin stream of water, and the retina, which becomes opaque a few hours after death, must be removed in the same way. The tapetum will then be fully exposed, and the light reflected from its surface

will be seen to have a very definite direction, adapted to the habits and instincts of the different classes of animals which possess it.

In the ox and in the sheep the tapetum is seen principally on the upper and outer part, in relation to the socket of the eyeball; whereas in the dog and the cat it is seen rather on the inner side.

The eyes in the ox and the sheep are placed on the sides of the head. In the lion, the dog, and the cat the eyes are placed more forward, and they can therefore use both eyes at once. In relation to the eyeball itself, the tapetum is found to occupy a different position in these different classes of animals. Taking the entrance of the optic nerve as a given point, the tapetum in the ox and in the sheep is seen principally on the upper and outer part. In the dog and the cat it is situated above the optic nerve and extends to about the same distance inwards and outwards.

The direction of the rays of light reflected from the tapetum is very remarkable. In the ox and in the sheep they are brought to an ill-defined focus; in the cat and the dog they are nearly parallel. This may even be observed without dissection. The reflection from the tapetum in a recently killed cat may be seen from the end of a room if the pupil be dilated. In the ox and in the sheep it can be best seen when the eye of the observer is near the animal's nose. When the anterior part of the eye, including the iris, is removed, the direction of the reflected rays becomes much more apparent, especially when the experiment is made some hours after the animal's death.

Experiment I.—In a calf's head, the roof of the orbit was taken away and the anterior part of the eyeball removed behind the ciliary ligament. The vitreous humour, some pigment which had escaped, and the retina were washed away. The tapetum was now seen, accurately and sharply defined, to occupy exclusively the upper and outer quadrant of the posterior half of the eye, with the

exception of a spur with a bulbous extremity which projected inward.[1] The tapetum had a bright metallic lustre, resembling mother-of-pearl. A light was now thrown on it in a room otherwise darkened, and the rays were reflected so as to be brought to an ill-defined focus about three inches to the outer side of the animal's nose. This focal concentration of light was very apparent on a black surface.

Experiment II.—A sheep's head was prepared in the same way as in the first experiment, and a light was thrown into the eye. The reflected rays from the tapetum were now found to come to the same kind of ill-defined focus, not on the side of the nose, but three or four inches in front of it.

In connection with these two experiments it is remarkable that the ox grazes from side to side; the sheep, forward.

Experiment III.—A cat was placed in a box with some chloroform. When it was dead, the pupils were found to be greatly dilated. The reflection from the tapetum could be seen in ordinary light from any part of a large room. It was visible, however, in one direction only, and that was in a line slightly diverging from the median plane laterally, and nearly parallel to the nose downward. The roof of the orbit was now removed and the anterior part of the eye taken away, as in the two former experiments. The reflection of light from the tapetum, which before was of a light yellow colour, now appeared of a very light green, of the brightest metallic lustre. The reflected rays of light did not here come to a focus as in the ox and the sheep, but were projected forwards and downwards, very much in the same direction as they were before the anterior part of the eye was removed. The tapetum was found to be situated altogether above the entrance of the optic nerve, extending nearly equally to its inner and outer side. It had a very sharp

[1] This spur varies in shape in different specimens, and is better developed in the sheep than in the ox.

well-defined outline, about the size and shape of a longitudinal section of a kidney bean, with its slightly convex edge upward. In relation to the orbit the tapetum appeared in great part on its inner side, and could only be partially seen from the median plane.

Experiment IV.—A young cat was chloroformed; the nictitating membrane and the eyelids being removed, the bright yellow glare from the tapetum was seen with nearly the same brilliancy from any part of the room. The cornea, iris, and lens were now removed, and the tapetum was seen in this instance to be of a bright yellow lustre. It was on the inner side of the orbit, so that, viewed from the median line, the whole of it could not be seen.

These experiments have been repeated in various ways, always with the same general results.

In the horse the tapetum is very well developed, and that part which is to the inner side of the entrance of the optic nerve is larger than in the ox. When the anterior half of the eye is removed and the vitreous humour washed out, the reflection from the two portions of the tapetum is seen of a very bright lustre. The light, however, from the two portions is not reflected in the same direction, nor can both be well seen at once. That from the outer portion is directed downwards and inwards, as in the ox—that from the inner portion, downwards and forwards.

The reflection from the outer portion is best seen at about a foot distant; that from the inner is seen clearly at the distance of six feet.

The tapetum in the horse when spread out measures fully two inches in its transverse diameter. From its extent the reflected light is thrown over a larger area than in the ox, and the rays are not parallel to each other as in the cat.

This extended reflecting surface, with a prominent and moveable eye, must give the horse the assistance of a considerable range of vision in twilight (or, as it appears to us, in the dark) from reflected light.

In dogs the disposition of the tapetum is very much the same as in cats. The lustre has appeared not so bright as in cats, but brighter than in the ox.

FIG. 1.

FIG. 2.

FIG. 3.

FIG. 4.

Figs. 1 and 2 represent the tapetum, *in situ*, in the two eyes of the same cat. They were necessarily drawn separately, as they could not be fully seen together without removing the nasal bones. Viewed from the front, where the whole of the tapetum could be seen, it appeared almost circular. The rays of light from the natural concave surface were reflected in nearly parallel lines within an area that would allow the whole to pass through a dilated pupil.

Figs. 3 and 4 represent the tapetum in two eyes of another cat, after they had been removed from their sockets. More of the anterior parts of the eyes had been removed than in those represented in Figs. 1 and 2. The tapetum in Figs. 3 and 4 is represented as it lay expanded on a flat surface. The transverse diameter therefore appears longer than it would in its natural concave position.

The ox and the sheep, having their eyes placed on the

sides of the head, see an object accurately with one eye only. The dog, the cat, and the lion having their eyes placed more forward, can use both at once. If the tapetum in the cat had been placed on the outside of the eyeball, as in the ox, the rays of light reflected from its surface would have fallen on the nose, or crossed each other immediately above it. In any case the reflected light could have been thrown on the same object from one eye only at the same time, and that in a different direction to that which is required by the animal's instincts. On the other hand if the tapetum of the ox had the same relative position as in the cat, it would be of little use as far as grazing is concerned.

The ox and the sheep have both very large pupils in the transverse diameter. All the rays of light reflected from the ground within a given area, which impinge upon the tapetum in these animals, are collected as from a concave mirror and again reflected in a concentrated form directly on their food. This provision must enable them to feed in the dim twilight with comparative comfort and safety from the admixture of foreign matter, alive or dead. The concave mirror situated within the eye itself acts the part of the concave mirror in an ordinary ear speculum.

In the daylight the pupil of the cat is often contracted to a mere line from above downward, but when the cat is excited the pupil becomes round and fully dilated. The glare of a cat's eyes when met in a dark passage has long been noticed, and from the fact that this is seen equally on both sides, it appears that the cat has the power of directing the reflected light from both eyes to the same object at the same time. The degree of convergence of the optic axes may therefore give the cat the power of estimating accurately the distance of its prey before making its spring.

The glare of the lion's eye was not unknown to Shakespeare. In his description of the dreadful night on the eve of the ides of March, he makes Casca say :—

"Against the Capitol I met a lion,
Who glar'd upon me."

The tapetum is situated in front of the choroid and may be dissected off it, leaving the choroid of its natural colour. This colour, according to Hunter, presents in different animals every shade from nearly white to black. A coloured choroid may therefore be mistaken for a true tapetum.

Among the eyes that I have examined I have found that the tapetum does not exist in the hare, rabbit, rat, sea-gull, heron, plover, rook, common fowl, landrail, moorhen, hawk, owl,[1] Egyptian vulture (*Neophron percopterus*), &c. The tapetum does not exist in the eyes of any fish which I have been able to obtain.

The conclusions arrived at are:—

1. That where the tapetum exists, the eye has, by reflected light, an illuminating power.

2. That this power can be utilised only at comparatively short distances, and that the eyes of fish and of birds (which have the longest and keenest vision) have it not.

3. That in animals which possess the tapetum the light reflected from its surface is directed in different classes of animals respectively in accordance with the wants and instincts of each.

[1] The eyeball of the owl is peculiar; it somewhat resembles a very small opera-glass. In common with the eagles and the hawks, the owl takes its food in its claws. Mice and rats generally take it in their fore paws; rabbits and hares eat deliberately and slowly, and masticate their food as they take it. None of these require the assistance of a tapetum either in catching their prey or in avoiding any foreign matter that might accidentally be mixed with their food.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' New Series, vol. ii, p. 50.)

ENTERIC FEVER AT SUAKIN,

WITH SOME

CASES OF MALARIAL-ENTERIC, OR TYPHO-MALARIAL FEVER.

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THE study of any outbreak of enteric fever is always a matter of interest, and when occurring under conditions differing widely from those found at home the interest is increased. We do not as yet know all about this disease. Wide differences of opinion will always exist as to its origin; and even the means by which it spreads are still subjects of controversy.

The majority of medical men in civil practice in this country, I believe, incline strongly to the opinion that enteric fever is due to a specific poison, and that its development in any individual must be from the absorption of a specific particulate poison resulting from some previous case of the same disease. With us the contamination of drinking-water is so frequently traced as the

carrier of infection that other modes of its diffusion run the risk of being slighted.

Among medical men in the army there seems to be a pretty general impression that enteric fever may, and often does, originate *de novo*, the aggregation of a large number of young persons in a tropical climate being quite sufficient to determine an outbreak of this disease, without any necessity for specific germs. Some authorities in the service, and medical officers who have served in India and in other tropical climates, incline to the view that there is more than one disease which produces the symptoms and the lesions which we are accustomed to consider peculiar to enteric fever. The results of my tropical experience, fortunately not very prolonged, as now brought forward, will, I think, serve to strengthen the view of specific infection, while they subvert the idea that contaminated drinking-water is its only mode of conveyance.

First as to the question of the cases of fever met with at Suakin being really enteric. Our Indian medical authorities point out some differences and difficulties in the way of diagnosis.

Dr. Gordon, C.B., late chief of the medical service in the Madras Presidency, is quoted by Sir J. Fayrer in his 'Croonian Lectures' (p. 173) to the effect that "if a non-specific fever in the tropics occurs in a young delicate lad, it will almost to a certainty become complicated sooner or later in its course by diarrhœa or dysentery; and ulceration will be found in the small or large intestines, Peyer's glands included. Is it meant," asks Dr. Gordon, "to call it 'enteric' in a sense that it is pythogenic? If so, I believe that the designation is wrong."

A probable explanation of the non-specific "enteric" fevers of tropical climates is given by Dr. Hall, of the General Hospital, Calcutta, who thus writes to Sir J. Fayrer ('Croonian Lectures,' 1882, p. 175):—"I believe that a large proportion of cases returned as typhoid fever have no right to that name. If a man die in India after having

an elevated temperature, and an ulcer can be found in his intestine, the case is at once called typhoid. But it takes a good deal more than an intestinal ulcer to make a typhoid fever. I have seen many cases that could not with any certainty be referred to any type of fever, but which had on the whole more resemblance to remittent than to any other, and which were found after death to be coincident with intestinal ulceration, but an ulceration distinctly not typhoid. It was an irregular ulceration by no means selecting the site of Peyer's patches, and very often encircling the intestine; and my experience is that this form of ulceration often occurs in cases that would better bear the name of 'remittent' than anything else." I shall presently refer to one case of this kind with no ulceration at all (Case 4). The setting in of the rains is stated to be the time for commencement of this non-specific "enteric" fever, and its spread is not due to contagion (Fayrer, loc. cit., p. 177).

It would be presumption on my part to pretend to decide, on the small experience of a four months' campaign, whether non-specific enteric fever is a reality or not; but the cases which came under my notice have some bearing on this question. I hope to show in this paper that the outbreak at Suakin was true enteric fever, that it could easily be accounted for on the specific theory of causation, and that instead of attacking first the young newly-arrived troops (represented by the Guards) it began with the seasoned troops who had been some time in hot climates. With regard to the spread of the disease, the peculiar nature of the water supply—viz. condensed—gives a special interest to this outbreak. Some of the cases will, I think, show clearly that the climatic conditions of the locality produced modifications of the disease not met with in England, and contribute something to the elucidation of the typho-malarial type of fever recognised in the Royal College of Physicians' nomenclature. My own cases, which may be classed as typho-malarial, are too few to affect the question whether they were enteric fever modified by

malarial influence or really a special and most dangerous idiopathic disease.

My appointment at the Base Hospital at Suakin from the commencement of the active operations in the Eastern Soudan this spring, enabled me to see much of the rise and spread of fever among the troops engaged in the expedition, and to collect some observations which I hope may be thought worthy of record. The Base Hospital was the largest hospital in the camp, containing accommodation for about 300 sick, each ward-tent having eight beds. Here was also the best place for observing what were the most prevalent diseases amongst the force, for almost all the more severe cases had to pass through this hospital. The system carried out in the arrangement of hospitals had for its object the prevention of overcrowding of the hospitals in the front, by the constant transference of patients to the rear. The Base Hospital was a kind of collecting station for all the troops, and the cases we had under treatment there would give a fair idea of the prevalent diseases amongst the whole force. By the courtesy of the other medical officers of the hospital I was able to observe cases in every division of the Base Hospital, and I was frequently asked to see medical cases in consultation with the officers under whose care these cases were. During the latter half of the campaign I had the charge of a division which contained eighty beds, and it was into this division that a large proportion of the cases of enteric fever were admitted. By these means I have been able to collect the temperature charts of some seventy cases.

Every precaution was taken to ensure a pure water supply to the troops, with no risk of contamination during its distribution. From the scarcity of water in the desert, the brackish quality of much of the well water that is found, and the risk of contamination of these sources from the filthy habits of the natives, it was necessary to supply the troops with condensed water. The supply of condensed water was continuous, and was

generally sufficient for all requirements. By the use of pure water thus secured for the troops, we might expect that diarrhœa and dysentery would be kept in check, but the admissions from these causes were considerable; for instance, the admissions to the Base Hospital for diarrhœa and dysentery for the week ending April 3rd, were 22 and 9 respectively; the week following they were 39 and 19, and for the next week the numbers are 39 and 15. These diseases occurred among officers and men alike, and certainly to some who never drank any but distilled water. It is evident therefore that climatic influence has much to do with the production of these ailments.

Suakin is built on an old coral reef; the pores of the coral are partly filled up with carbonate of lime, and in some parts converted into solid blocks like marble. The interstices between the coral are filled up with sand, and the whole covered with a layer of loose sand like that on the desert beyond. The coral extends about three quarters of a mile inland, and then the foundation becomes rock. The Base Hospital was on the coral, as were all the camps at the commencement of operations. After a time some of the camps were moved on to the rocky ground towards Handoub.

After March the temperature during the twenty-four hours ranged about 80° F., the average daily variation being 23·5°; the greatest variation being 35°. The air was dry, with heavy dews towards sunrise; the prevailing wind N.E., comparatively cool, with occasional hot southern winds.

Thus we see that the soil was porous and quickly absorbed moisture, and the rapid desiccation of excreta and refuse from the heat would favour the dissemination of particles into the air. The falling tide left dry much of the shallow lagoons which bordered the deep harbour, giving a broad stretch of damp ground covered with excreta. Here the native population always resorted to the shallow water for the act of defæcation. The early morning dew gave moisture enough for the existence of

malarial germs, while the heat was the cause of many men being invalided from exhaustion and sunstroke. The great variations in temperature were also trying to men used to a more temperate climate and were probably the cause of the attacks of acute tonsillitis that occurred. With regard to malaria, the general impression amongst the army surgeons seemed to be that Suakin ought not to be considered a malarious situation.

Very few cases of ague were admitted into the Base Hospital. I had only two or three in my division, and these occurred in men who had previously suffered from malaria in India. But there was a form of fever which attacked men who had never been in any malarious district, which, as far as my personal experience went, began usually about 5 o'clock in the afternoon with a feeling of soreness all over the body, with headache and a tendency to giddiness and slight elevation of temperature (100° to 102° F.). This had completely disappeared by next morning, but returned in the evening. After two or three attacks it might show itself in the morning and persist all day. During one such attack my temperature was 102° F. before noon. I could detect no periodicity in the attacks, which have since recurred, but I found large doses of quinine of great service. I think it likely that these feverish attacks may be really of malarial origin.

Before commencing to discuss more particularly the outbreak which occurred among our troops, it may be as well to give what proofs I am able that the disease was in reality enteric fever. In the first place the symptoms were in every respect similar to those which are seen in enteric fever in this country. Of course many cases showed modifications, and there were some in which the diagnosis remained doubtful for part or the whole of the illness. But there was a sufficient number of cases which presented symptoms which left no doubt as to their nature.

The onset was gradual, the men usually being admitted after a few days' illness, with increased temperature and diarrhœa. The tongue in some cases was typi-

cally dry and brown, the stools presented the ochre colour or light brown "pea-soup" character; and splenic enlargement with tenderness in the right iliac fossa and gurgling were present. A difficulty was found with regard to the recognition of the specific eruption, in that the body was often spotted with sudamina, which were generally most abundant over the abdomen in consequence of the flannel belt which all the men wore night and day. Some cases showed no sign of the specific eruption. The progress of the cases also resembled that of enteric fever in this country, and the temperature charts will be seen to show similar curves to those met with here. In order to completely satisfy myself of the nature of the disease I made a small number of post-mortem examinations; but the rapidity with which decomposition set in, and the discomfort of making autopsies on the floor of a bell tent with the temperature above 90° F., caused me to confine the examination to the intestines.

The first autopsy was in the case of Private J. H—,[1] æt. 24, 2nd East Surrey Regiment, who was taken ill about the 20th of March. He was admitted to hospital on the 31st, and died on the 18th of April, or about the thirtieth day of illness. Post mortem there was found much ulceration of Peyer's patches in the lower part of the small intestine, the ulcers having the undermined edges and other characters of ulcers in enteric fever. There was also some hypostatic congestion of the lungs. No perforation of the intestines had occurred. The diarrhœa in this case was to the extent of about four to six stools daily, but the temperature was most irregular, running up unexpectedly three or four degrees and coming down as suddenly as it rose. The highest temperature was 104·6° F. (Chart I).

The next case is that of Private J. G—, East Surrey Regiment, æt. 23, who was taken ill on April 7th, and was admitted on the 14th. Rose spots were noticed on the tenth day, and the patient died from exhaustion on the thirteenth

[1] See Temperature Chart, Pl. VII, fig. 1.

day. The temperature was persistently high during his stay in hospital, ranging from 103° to 105° F. *Post mortem* infiltration of Peyer's patches was found, and great enlargement also of the solitary glands.

Many of the cases went through the disease without any marked deviation from the typical course of enteric fever.

I have records of seventy-three cases which I believed to be enteric fever. Of these, forty-four were under observation for the whole illness, and twelve died; and the remaining thirty-two were sent to England convalescent. Of the twenty-nine cases which had to be sent out of the Base Hospital before they could be fairly said to be convalescent, nine had been ill for more than three weeks, and in most of these the temperature was gradually coming down and they were nearly convalescent. Another nine had been ill over two weeks, and all but two of these were improving satisfactorily. Seven more had been under observation less than a fortnight, of these three had only been in hospital three or four days and no certain diagnosis was possible. There remain three cases which I have kept separate because of the diagnosis put against them. Serg. E—, æt. 34, of the Medical Staff Corps, was diagnosed "febricula"; Private S—, æt 25, 5th Lancers, was diagnosed "simple continued fever"; and Sapper Mc N—, R.E., æt. 26, is entered as "sunstroke." Yet on comparing the temperature charts of these cases with those of undoubted cases of enteric no great difference will be seen, and I suspect that enteric fever would have been a truer diagnosis.

In looking over the complications that were met with during the course of the cases, we find epistaxis with hypostatic congestion occurring both early and late in the disease in several cases, and in one case there was the rusty expectoration of pneumonia, but without any stethoscopic signs. Vomiting of bilious matters occurred in three cases. These cases, where vomiting was a prominent and early symptom, died, thus illustrating the seriousness of such cases. In 'Quain's Dictionary of Medicine' they are referred to as "Bilious Typhoid." Involuntary micturition

occurred in some of the more severe cases, while retention was noticed in two.

Private H—, æt. 22, a Mounted Infantry man, who was ill for thirty-one days without his temperature falling to the normal, had at last the whole body covered with sudamina, every square inch, including the face, being thickly studded. During the last week or ten days before he was transferred, a bedsore had formed on the shoulder; but this was the only bedsore I observed in the hospital.

Hæmorrhage from the bowel only occurred in two of my own cases.

Private E—, of the Grenadier Guards, seems to have had some peritonitis on admission, and to have died from perforation. There was no autopsy.

Five of the more serious cases are, I believe, instances of "typho-malaria," or more strictly "malarial enteric Fever," and these I will now give more fully.

Case 1 is Mr. R—,[1] a correspondent, æt. 25, whose duties involved a good deal of exertion. He was laid up for a few days in the beginning of April, but resumed his work. On April 27th he was again admitted with diarrhœa and a temperature of 103·6° F. in the evening. He said he had then been ill three days. The temperature remained constantly high—between 102° and 104° F., and he was much depressed. Soon great restlessness and anxiety came on, followed by delirium, at first only at night, but finally by day as well. Soon after admission a large purpuric blotch about the size of the hand, like a big bruise, was noticed on the left forearm, and later others appeared on the legs and trunk. He gradually sank, and died on May 15th, the twenty-first day of the disease. In this case the probability of scurvy seems excluded, as Mr. R— was able to get every variety of diet, and lived well, either in the town or in the Head Quarter Camp, until he was taken ill.

A similar appearance was seen in Case 2. Private E—,[2]

[1] For Temperature Chart, see Pl. VII, fig. 2.

[2] For Temperature Chart, see Pl. VII, fig. 3.

æt. 22, Grenadier Guards, was admitted on the 10th of May, having been ill five days. His temperature on admission was 103·4° F., but rose the next evening to 105·6° F., and he was delirious at night. On May 13th—the eighth day of illness—some purpuric spots were noticed, which on the eighteenth had increased, till the condition at that time was as follows. The upper and lower eyelids of both eyes were purple, giving the appearance of ordinary "black eye" from a blow. There was subconjunctival hæmorrhage on the inner half of both eyes, causing the conjunctivæ from the pupil to the inner canthus to be bright red. Other spots and blotches also appeared on the arms and trunk. This patient died on May 22nd, the seventeenth day of illness. Here it may be noted that oranges were given to the troops when possible, and lime juice was also served out, and as no scurvy was noticed amongst the troops it is impossible to consider the appearance noticed in these two cases as due to that condition. In further considering the cases with a view to see if any malarial influence can be detected in any of them, I find one which presents a markedly remittent character (Case 3). This patient, Private M—,[1] æt. 21, of the Shropshire Regiment, was admitted on April 20th, his temperature that evening being 103·8° F. The next day he had vomiting and abdominal pain, with a temperature of 105° F., and a pulse of 108 per minute. The general symptoms led to a diagnosis of enteric fever being formed. On the 24th of April—after four days' illness—the temperature fell rapidly till it reached, on the next day, 99° F. He was now feeling much better, and being able to answer questions gave the following history. He had been stationed with his regiment in Malta, and during a severe epidemic of enteric fever in the island in the summer of last year—in which his regiment alone lost forty men—he was taken ill, and admitted to hospital there on November 27th. He was in hospital for three months, suffering from enteric fever, and when the regiment left

[1] For Temperature Chart, see Pl. VIII. fig. 1.

for the Soudan he was still unfit to accompany it. He followed, however, on March 21st, arriving at Suakin about April 7th. Soon after passing Suez his diarrhœa commenced again, and continued till his admission to the Base Hospital. On April 18th, whilst on duty guarding the railway, he became giddy and had to go back to camp, whence he was brought to the hospital next day. On the 21st the motions were liquid and light coloured, having the character of enteric fever stools. The temperature, after remaining low for two days, again rose on the 25th and remained high for three days, falling again till it reached 97·6° F. on the 1st of May and then rising again. Here it is probable that the remissions were due to *malarial influences to which he had been subjected in Malta.*

This case may, I think, fitly be classed as *malarial enteric.* It will be noticed that the patient had been in hospital for three months in Malta with an illness that was there described as enteric fever. His attack at Suakin certainly was not a relapse, and second attacks of enteric fever are rare. The malarial influence in this case is incontestable, and I can answer for the existence of enteric symptoms—sometimes absent in cases published as typho-malaria.

There now comes a case (4) which I must mention on account of the symptoms resembling these entero-malarial ones, and from the autopsy showing us an unexpected state of the alimentary canal.

Private J—,[1] æt. 25, Coldstream Guards, was admitted on April 24th with an evening temperature of 102·8° F. He was a big, florid man, and the symptoms led to the diagnosis of enteric fever. On April 27th, the ninth day of illness, there was vomiting which recurred frequently up to the time of his death. On the tenth day of illness rose spots were seen on the abdomen. On the twenty-fifth day of illness he died rather suddenly about 2 p.m., and I made an autopsy the same afternoon. Instead of finding, as we expected, extensive ulceration of the lower

[1] For Temperature Chart, see Pl. VIII, fig. 2.

ilium, we could not discover a single ulcer; while the whole ilium showed marked injection of the vessels, with hæmorrhagic spots in the mucous lining of the intestine.

This case at once brings to our mind the two cases (Private E— and Mr. R—,) in which subconjunctival and cutaneous hæmorrhages were found, and suggests that possibly post-mortem examination in those cases also might have shown a modification of the lesions in the alimentary canal. These cases in fact cannot easily be referred to any type of fever with which I am familiar. Typhus may be at once excluded; for not only were the petechial extravasations entirely unlike the mottled marks of this fever, but these cases were apparently in no way contagious.

How far the heat of the climate was concerned is worthy of consideration. Heat alone can hardly have been the only modifying cause, and is not likely to have produced the illness. Cases of typical sunstroke and heat apoplexy were comparatively rare; and among the very numerous cases of heat exhaustion the temperature was almost always low, and only in one or two cases rose to over 100° F. In most cases of this kind recovery was rapid, and no petechiæ were noticed. In the fever of tropical acclimatisation diarrhœa is not, I hear, a prominent feature, as it was in this case.

Such fevers attack those who arrive in India during the hot season, when perspiration is checked by the moist air of the monsoon; this is not of malarial origin, for it has no intermittent character, and does not recur. As far as I am aware hæmorrhagic patches, such as I have described, are not met with in these cases, nor is vomiting a frequent or persistent characteristic. Heat alone will produce diarrhœa, but it is a diarrhœa not attended by fever; in fact in some cases of simple diarrhœa in which I took the temperature it was rather subnormal, as it was also in one or two cases of simple catarrh (cold in the head). Heat and chill may be important factors in the production of dysentery, but these cases have no resem-

blance to that. The possibility of some malarial influence in this case is suggested by an intermittent character in the temperature chart. To use the term "bilious remittent" is to beg the main question. To me it seems that enteric fever is chiefly indicated, but modified either by some malarial or by some climatic causes. If we have not in these cases some disease which cannot be referred to any of the classes usually recognised and described, but merely enteric fever modified by climatic conditions, the modification which will cause the absence of the ordinary ulceration of the intestine, even after three weeks' illness, as was the case with this patient, is one of unusual importance.

In connection with these cases, I must mention one (Case 5[1]) which occurred during the voyage home. The patient was one of the railway navvies, a big powerful man of about thirty years of age. He reported himself sick with headache and diarrhœa on May 28th, soon after leaving Suakin. It was first supposed to be merely indisposition from the heat, but the diarrhœa continued and the illness became more marked. We had left a man behind at Suakin who had been ill on board with enteric fever, and had already two or three other cases in the ship which looked like the same disease. This man, S—, was ill all the voyage, and I almost despaired of his reaching England. He appeared to me to be suffering decidedly from enteric fever, but the temperature chart shows in a marked degree the large variations which I attribute to a malarial influence. During the course of his illness he got some pulmonary complication, which, however, was not severe. This was in the second week. On several occasions I gave him quinine, but never in sufficient quantity to have any marked effect on the temperature. Diarrhœa was a marked feature of the illness, and the prostration was extreme.

He recovered after an illness of about thirteen weeks. If this man had been left at Suakin I believe that he

[1] For Temperature Chart, see Pl. VIII, fig. 3.

would not have recovered, and that the removal into a healthy climate gave him his only chance.

The long continuance of this case and that of Case 3, both with recovery, and the absence of enteric lesion in Case 4, suggest the question whether the special processes of enteric fever may be modified by malaria; and again, may not Cases 1, 2, and 3 be really not cases of enteric fever at all; and the two cases of "bilious typhoid" be partly owing to malaria? It would appear that the existence of undoubted enteric fever does not necessarily prevent a lowering of temperature to nearly 99° F. in the first week, when there is no reason to suspect any malarial influence; and that in those cases where convalescence is prolonged, it is the evening rise, rather than the morning fall, which characterises the irregularity.

I will add a short analysis of my cases and a few words in conclusion on the origin and spread of the epidemic at Suakin and on some points concerning the etiology.

The following table shows the number of cases of which I have records. It will be seen that the majority went through the whole illness under observation at the Base Hospital; and of the uncompleted cases the majority were in a fair way towards convalescence when they were transferred to the hospital ships.

Completed Cases (44)	Died	12
	Convalescent. . .	32
Uncompleted Cases (29)	Over 3 weeks ill . .	9
	Over 2 weeks ill . .	9
	Under 2 weeks ill . .	7
	Probably enteric but variously named . .	4
	Total	73

Deaths = 12 *in* 73, *or about* 1 *in* 6.

Although this number of deaths cannot be taken as the whole mortality for the 73 cases, I think it will not be very far short.

In looking at the ages of those who came under my

notice for enteric fever, with a view to discover if age influenced the mortality, I find that of the 73 cases there were

2 cases with no deaths over 30 years of age.
22 „ 5 „ 25 „ „
39 „ 6 „ 20 „ „
4 „ 1 under 20 „ „
And in 6 cases the age is not stated.

It must be remembered that by far the majority of the troops were from 20 to 25 years of age and very few were over 30.

From the following table it will be seen that, though more cases occurred among the younger men, the mortality was greater among those over 23 years of age than in those below that age.

Age.	Cases.	Deaths.	Mortality.
19 to 23 . .	36 . .	5 .	1 in 7·2
24 to 27 . .	26 . .	7 .	1 in 3·7
28 and over .	5 . .	0	
Not stated . .	6 . .	0	

Or, to divide them differently,

23 years and under, 36 cases with 5 deaths, or 1 in 7·2.

Over 23 years, 31 cases with 7 deaths, or 1 in 4·4.

The disadvantages of youth in this disease, on which so much stress is laid by the Indian and other army medical officers, is not therefore apparent in these cases.

Nor did the new-comers suffer most; the seasoned regiments furnished some of the earliest cases.

At Suakin the first dozen cases admitted into the hospital came from the Marines, the East Surrey Regiment, and the Commissariat Corps—chiefly from the Surrey men —and cases had been admitted from all these corps a full fortnight before any men were admitted from other regiments. Of these corps the Marines and Commissariat had had men at Suakin for the previous twelve months, and the East Surrey came straight from Cairo, where there is

always enteric fever to be found, having been stopped on their way home after several years in India. Thus, they all had spent some months in the country before the Guards arrived in March. As the Guards came straight from England it was to be expected that, if the "aggregation of young soldiers in a tropical climate" is sufficient to start an epidemic of enteric fever which "chiefly attacks the new-comers" (Sir J. Fayrer, 'Cr. Lect.,' p. 176) the Guards would have furnished the early cases. As a matter of fact, however, no case occurred in the Brigade of Guards till three weeks later than the first cases in all the regiments mentioned above and not until they had been six weeks at Suakin.

The question as to which regiments supplied the first cases of enteric fever is of importance as furnishing a guide to the origin of the epidemic. It is time enough to be content with a theory of spontaneous origin when we can find no trace of a cause which will satisfy the more generally accepted specific origin of the disease. But here, I think, we shall have very little trouble in tracing the epidemic to pre-existing enteric fever elsewhere. Of course when once introduced the disease spread rapidly.

The regiment which furnished the first cases was the East Surrey Regiment. They arrived at Suakin from Cairo, about February 20th, and on March 31st they had a patient admitted to the Base Hospital, suffering from enteric fever (Private J. H—). In this case the nature of the disease was not open to doubt, as the result was fatal, and the autopsy showed typhoid ulcers in the intestine (see page 253). This is the first case in point of time, and we find that at Cairo enteric fever existed at the time when the regiment left. There is another point about this regiment which deserves notice. Early in March they were encamped to the northward of Suakin and for the first three or four days of their being there drank well water, until a tank was placed for them to keep a supply of condensed water. This is the only instance I heard of men drinking well water. The first case from the

Berkshire Regiment was admitted on April 14th, though they arrived at Suakin in January. The first case from the Shropshire Regiment was admitted on April 20th; from the Guards Brigade on April 24th, and from the Cavalry on April 25th, which allows sufficient time for all of these to have become infected from the East Surrey Regiment, which had sent a man to hospital with enteric fever a fortnight before. As regards the Australians they had a case of enteric fever on board when they arrived at Suakin, the man having been taken ill soon after passing Aden.

Perhaps the most interesting point, however, with regard to the etiology of enteric fever which this epidemic presents is connected with the spread of the disease. The care taken to prevent the men drinking contaminated water, by the constant supply of condensed water, makes it almost impossible that the disease could have been propagated by the drinking-water in the manner so frequently looked at as the chief mode of infection. The most natural inference from a consideration of the circumstances is that the infection was conveyed by the air; and strong probability exists in my opinion that it was by this means that the disease spread. All the camps had latrines formed by digging a trench about two feet deep and two feet wide, into which all the excrement was passed. From the heat of the sun this was soon dried, and pulverised particles could easily be carried by the wind. Defæcation was by no means limited to the trenches prepared for the purpose. From the cases I have quoted it will be seen that most of the patients had been ill some days before admission to hospital; and in one case (Private G. H—, æt. 24, Berkshire Regiment) the patient, who was a mess orderly at the Head Quarter Camp, had been ill for three weeks before he reported himself sick. Until admission to hospital these men would use the common latrine, perhaps sitting beside some other man or being immediately followed by one who might place himself directly over the source of infection. When it is remembered that soldiers

have a peculiarity of remaining for a considerable time on the latrines, so much so that in one military hospital in Egypt I saw a sentry placed over the latrines with orders to turn any man out who remained as much as an hour—it would seem possible that infection might be caught in the latrine. Another fact in favour of infection having taken place by particles in the inspired air is seen in the large number (nine) of the Medical Staff Corps orderlies who were attacked. Nearly all these men were on duty at the Base Hospital and in charge of fever tents; and whereas the earliest case from them was admitted on April 24th, by which time we had over a dozen cases in the hospital, the majority of the cases were not admitted till the second week in May. These orderlies performed all the duties of nurses to the patients, including, of course, the removal of the bed-pans. They were also expected to wash, and soak in disinfectants, all soiled linen from the fever cases before sending it to the laundry. They were very hard worked, many of them never getting more than six hours off duty at a time for six weeks, and in some cases they had, like all the other tent orderlies, to sleep on the ground in the tent for which they were responsible. They were thus constantly exposed to the air contaminated by the exhalations from the patients; though they were not allowed to drink anything which had stood in the fever tents, or even to use for themselves the water from the filters in those tents. The constant visits of the Sisters and medical officers to the tents acted as a check on infringement of these orders.

It may of course be urged that infective particles might have been carried in the air and settled in the water which was stored in the tanks for the use of the troops; but this was hardly possible as the tanks were usually carefully covered with an iron lid.

In reference to the probability of the infection being taken in by the inspired air, I am reminded of a case which occurred to me when I first went into residence at University College Hospital as house physician in 1881.

Two or three of the attendants in one ward which I took over had been attacked with enteric fever, and I reported the ward sinks as unsatisfactory. On examining these it was found that the special sink, for emptying the contents of the bed-pans into, was choked at the trap, and that the dejecta consequently lodged there. There were cases of enteric fever in the ward and the stools were emptied down this sink, and it seemed then that the nurses might have contracted enteric fever from inhaling the exhalations from matters blocked in this pipe and rising into the scullery.

This outbreak of enteric fever at Suakin is thus interesting from its bringing out the following points:

1. The disease was imported. The infection was brought from Cairo and no theory of spontaneous origin is necessary.

2. It spread by infection, the medium of transmission of the infection being the air. The use of condensed water for all drinking and cooking purposes made transmission by the water almost impossible, and thus makes the history of this epidemic a valuable addition to our facts on the mode of conveyance of enteric fever infection.

4. The mortality was not proportionally greater in the younger men, although the majority of those attacked were young. The troops engaged were mostly young.

5. The climatic conditions produced in some cases modifications of the disease, which seems to justify the term *malarial enteric fever*.

6. In addition to a modified form of enteric fever, there would seem to be justification for the term *typho-malarial*, as applied to cases (such as Case 4) in which no typhoid ulceration is found after death.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' New Series, vol. ii, p. 52.)

DESCRIPTION OF PLATES VII AND VIII.

(Enteric Fever at Suakin. By J. EDWARD SQUIRE, M.D.)

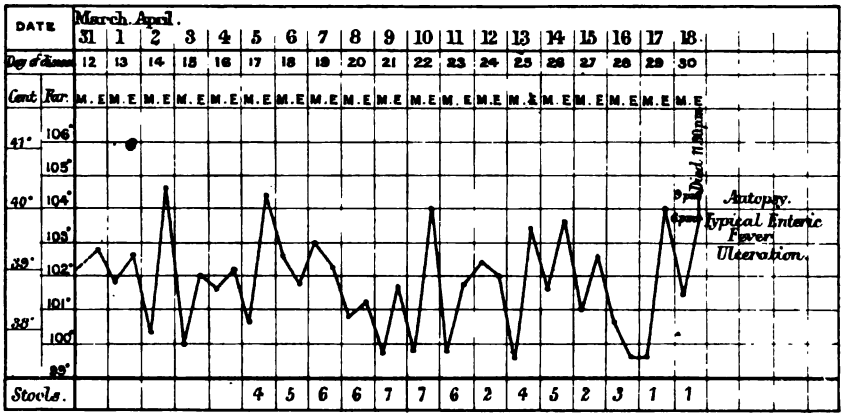
PLATE VII.

Fig. 1.—Temperature Chart.—J. H— (see page 253).
2.— „ Mr. R— (see page 255).
3.— „ Private E— (see page 255).

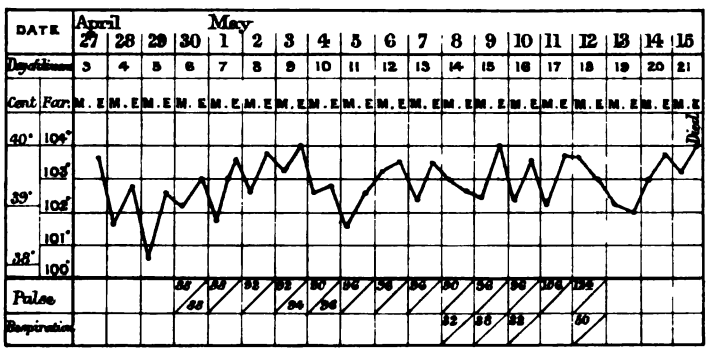
PLATE VIII.

Fig. 1.—Temperature Chart.—Private M— (see page 256).
2.— „ Private J— (see page 257).
3.— „ S— (see page 259).

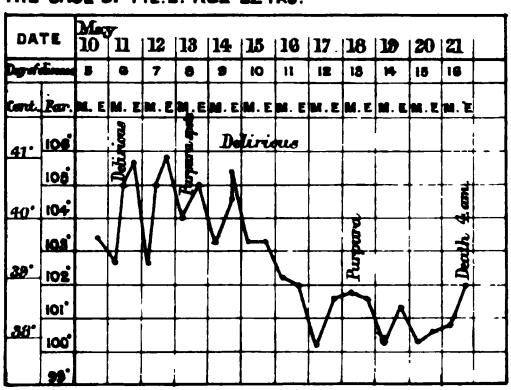
THE CASE OF PTE. J. H. AGE 24 YRS.


DATE
March. April.
31 1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18
Day of disease
12 13 14 15 16 17 18 19 20 21 22 23 24 25 26 27 28 29 30
Cent. Far.
M. E M. E M. E M. E M. E M. E M. E M. E M. E M. E M. E M. E M. E M. E M. E M. E M. E M. E M. E
41° 106° 105° 40° 104° 103° 39° 102° 101° 38° 100° 99°
Died 11.30 p.m.
Autopsy. Typical Enteric Fever Ulceration.
Stools.
4 5 6 6 7 7 6 2 4 5 2 3 1 1

THE CASE OF MR. R. AGE 25 YRS.

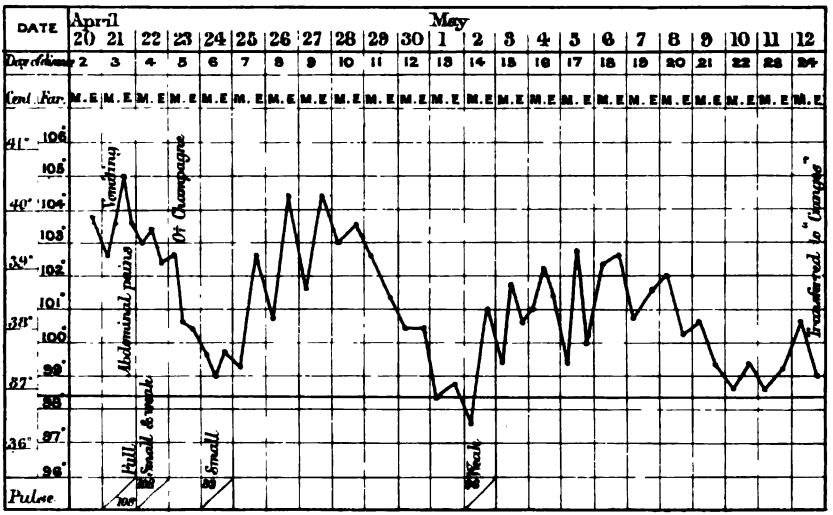


THE CASE OF PTE. E. AGE 22 YRS.

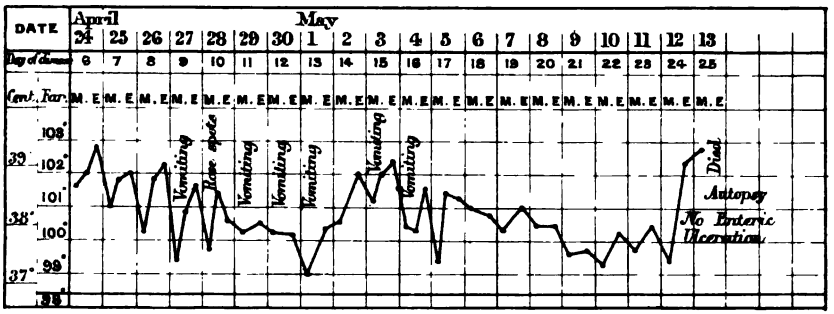

DATE
May
10 11 12 13 14 15 16 17 18 19 20 21
Day of disease
5 6 7 8 9 10 11 12 13 14 15 16
Cent. Far.
M. E M. E M. E M. E M. E M. E M. E M. E M. E M. E M. E M. E
41° 106° 105° 40° 104° 103° 39° 102° 101° 38° 100° 99°
Delirious
Purpura appears
Delirious
Purpura
Death 4 a.m.

Mintern Bros. lith.

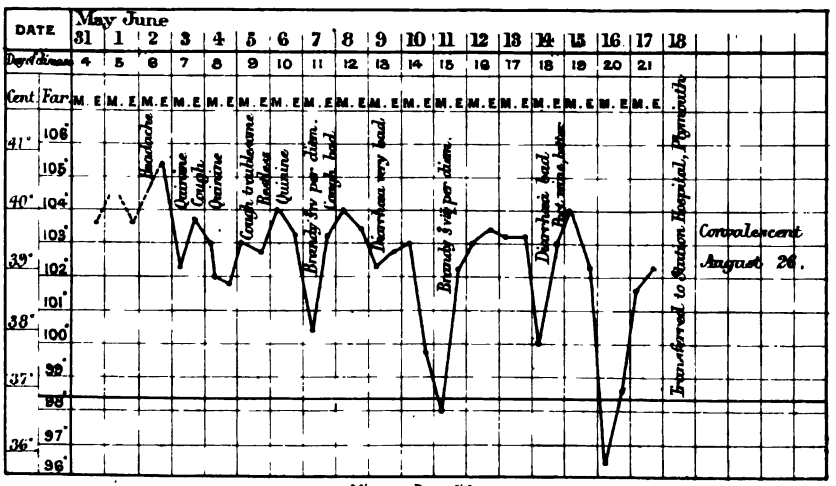
THE CASE OF Pte. J. M. AGE 21.



THE CASE OF Pte. J. AGE 25.



THE CASE OF S.



Mintern Bros. lith.

A CASE OF THORACIC ANEURISM

TREATED BY THE

INTRODUCTION OF STEEL WIRE INTO THE SAC.

BY

WILLIAM CAYLEY, M.D.,

PHYSICIAN TO, AND LECTURER ON THE PRINCIPLES AND PRACTICE OF MEDICINE AT, THE MIDDLESEX HOSPITAL; PHYSICIAN TO THE FEVER HOSPITAL AND TO THE NORTH-EASTERN HOSPITAL FOR CHILDREN.

Received December 8th, 1885—Read February 23rd, 1886.

THOS. B—, æt. 48, a publican, was admitted into the Middlesex Hospital, June 5th, 1885, under the care of Dr. Cayley.

Patient, who was formerly a sailor, had not had good health for some years. He had been subject to rheumatic gout, and what he described as liver complaint, and had probably been a pretty free drinker. In 1858 he had syphilis, and in 1860 an attack of fever at Calcutta.

In November, 1884, he began to suffer from symptoms of thoracic aneurism, and attended the Middlesex Hospital as an out-patient under Dr. Fowler, but it was not till five days before his admission that a visible tumour made its appearance at the root of the neck.

On admission, he was a well-nourished man of good muscular development and rather florid complexion. He complained of a constant aching pain over the upper part of the chest, which prevented sleep; he had a clanging

metallic cough with inspiratory stridor, and there was some difficulty in swallowing.

There was an oval elastic swelling about the size and shape of a hen's egg, situated above and behind the right sterno-clavicular articulation, which was bulged forwards; the tumour rose about three inches into the neck and encroached upon the sternal notch. It had a very distinct expansile pulsation, and on auscultation the heart-sounds were very plainly audible over it; but there was no bruit. There was dulness on percussion over the tumour and for some distance below it over the sternal region. The heart-sounds were normal, but the heart was a little displaced downwards and to the left. There was no difference in the radial pulse on the two sides; the pupils were equal and normal. The air entered both lungs equally; the breathing was attended by much tracheal stridor. Pulse 84, resp. 20, temp. 98·4°.

He was directed to keep constantly in the recumbent posture, and was ordered a diet consisting of milk six fluid ounces, beef tea six fluid ounces, meat five ounces, and bread five ounces, with two eggs, and he was given twenty grains of iodide of potassium three times daily; this was gradually increased to sixty grains three times daily. He was also given opium to procure sleep.

Under this plan of treatment the tumour rapidly increased, and it was evident that it must soon burst externally or become diffused among the tissues of the neck.

A consultation was held with my colleagues, and it was decided to treat the aneurism by introducing wire into it, as was practised by the late Mr. Charles Hewitt Moore in a case published in vol. xlvii of the Society's 'Transactions.'

I was induced to urge this course from having made the post-mortem examination of Mr. Moore's case, which satisfied me that the fatal termination was due entirely to pyæmia, the result probably of some septic poison having been introduced into the sac. So far as the aneurism was

concerned the operation had been successful, consolidation having been effected.

In the present case there was some doubt as to the point of origin of the aneurism, whether it might not spring from the innominate artery and so be amenable to distal ligature. Aneurisms of the innominate are, however, very rare as compared with those of the arch; in this case, too, the carotid artery seemed to come from behind the tumour; moreover, Dr. Fowler, who had treated the patient at the onset of the symptoms, then considered it to have been aortic.

My own opinion was that the introduction of wire into the sac was under any circumstances a less dangerous proceeding than distal ligature of the subclavian and carotid arteries.

Mr. Hulke having concurred with me that this operation would, under the circumstances, give the patient the best chance, it was accordingly determined on.

A long coil of fine steel wire, prepared by winding it in a very close spiral on a mandril, was cleaned by placing it for twelve hours in strong Liquor Potassæ in order to remove adherent grease and render it antiseptic.

I believe that steel wires are liable to have adherent to them grease which is used in tempering them, and this might readily be the means of introducing septic material, and to this, or to the trocar and cannula not having been thoroughly disinfected, I am disposed to attribute the occurrence of pyæmia in Mr. Moore's case.

The coil of wire thus cleansed was placed on a brass cylinder of a half an inch in diameter. The spirals were consequently very small, and though this rendered the introduction of the wire more troublesome, it had the advantage of causing it to coil up in the neighbourhood of the puncture instead of passing to a greater distance in indeterminate directions.

On June 28 the patient was anæsthetised and Mr. Hulke introduced a fine trocar and cannula, the lumen of which was just sufficient to easily transmit the wire, into

the aneurism an inch above and a little to the outer side of the right sterno-clavicular articulation, and after withdrawing the trocar passed forty feet of the wire through the cannula into the sac. Strict antiseptic precautions were observed, including the passage of the wire, as it was drawn off the cylinder, between two folds of sponge wet with a 2½ per cent. solution of carbolic acid.

On withdrawing the trocar a little blood spurted from the cannula, and during the passage of the wire some oozed by its side. When the cannula was withdrawn a localised hæmatoma formed under the skin.

No constitutional disturbance followed the operation; the temperature remained normal and the pulse unaffected. The pain at the root of the neck, of which he had previously complained, much abated. The following day it was noticed that the pulsation of the tumour was much less marked.

The hæmatoma gradually absorbed, and the tumour became converted into a hard mass with a slight communicated pulsation. The clanging cough, laryngeal stridor, and occasional attacks of dysphagia continued.

On July 3rd an irritable pustular rash appeared on the thighs, due probably to the iodide of potassium; this was accordingly discontinued.

In August signs of extension of the aneurism to the left and backwards showed themselves.

August 9th.—It was noted that the tumour on the right side was quite hard and free from pulsation. To the left of the tumour in the sternal notch, and behind the left sterno-clavicular articulation there was distinct pulsation. There was dulness on percussion over the upper part of the sternum reaching an inch to the left of its left border. Over this region the sounds of the heart were abnormally distinct, but there was no bruit.

There was much stridor with the breathing, and frequent attacks of coughing accompanied by severe dyspnœa, during which the face became much congested. Some glairy mucus was expelled with great difficulty.

It was now evident that the aneurism would soon prove fatal from pressure on the trachea, and I thought it might be possible to cause consolidation of that part of the sac which was causing this pressure by a repetition of the operation.

Accordingly, on September 10th, the patient was anæsthetised, and Mr. Gould, in the absence of Mr. Hulke, who was away for the autumn vacation, introduced a trocar and cannula into the sac above and to the left of the left sterno-clavicular articulations, directing the instrument somewhat obliquely inwards towards the middle line, and passed in thirty-four feet nine inches of wire. At first the wire met with some resistance, but this soon ceased and it passed easily. At the end resistance was again felt and the wire was then cut short and the end pushed down the cannula into the sac by passing a blunt trocar. About a fluid drachm of dark blood escaped during the operation. Some hours after the operation the patient vomited after taking food. At 5 p.m., eight hours after the operation, the temperature was 99·4°; at 10 p.m., 98·2°. He passed a pretty good night, but coughed several times and had difficulty in expectorating. During the next four days he continued much in the same state as before the operation, still having attacks of cough and dyspnœa; the temperature varied from 99·4° to 98·2.° He also continued to complain of a good deal of pain over the upper part of the chest, which was perhaps more severe than before the operation.

The pulsation over the upper part of the sternum and to the left of it did not appear to be much altered.

September 16th.—He complained of a good deal of pain about the region of the last puncture, passing through to the shoulder and down the left arm. The attacks of coughing and dyspnœa were more severe and frequent. Temperature continued to fluctuate between 99·4° and 98·6.° It was thought there was some increased pulsation over the aneurism. An ice-bag was applied and morphia administered subcutaneously.

The next day the pain was less, but the temperature

rose at night to 102·4. The attacks of cough and dyspnœa continued to recur.

September 18th.—The temperature had fallen to 98·2°.

September 19th.—Temperature at 10 a.m., 97·6°. In the evening he was seized with a severe attack of dyspnœa, during which his face became congested and cyanosed. He was given a hypodermic injection of morphia, but without relief. He became unconscious, with twitchings of the muscles of the face and limbs, and died in about two hours.

On post-mortem examination a large aneurism was found to spring from the ascending part of the arch of the aorta, which was generally dilated and atheromatous. The aneurism, which communicated with the artery by a very large opening, extended up behind the sternum into the neck, reaching on the right side to three inches above the sterno-clavicular articulation. The walls of this part of the tumour were only formed by a little condensed connective tissue about the sixth of an inch in thickness. The upper part of the sac was filled by a firm pinkish clot, embedded in which were the two coils of wire. Below this for some distance the cavity was occupied by softer blackish clot; then there was a layer of decolorised, fibrine which separated this part of the sac from the lowest part which was in immediate continuity with the dilated vessel. This lowest part of the sac contained no clot, and corresponding to it the trachea, a little above the bifurcation, was flattened by pressure and its mucous membrane much reddened and inflamed. The bronchial tubes and the lower part of the trachea contained much viscid mucus. The upper part of the sternum was eroded and its inner surface exposed in the aneurismal sac. The heart itself was normal. The branches of the arch were not implicated in the aneurism.

The other organs presented nothing abnormal. No embolisms were discovered.

In this case the first operation produced the desired effect of consolidating that part of the aneurism which

projected into the neck, the rupture of which was imminent. The second operation only completed the consolidation of the upper part of the sac, but had no effect on the lower part which was compressing the trachea. This part of the sac communicated with the aorta by so large an opening, and was in such immediate connection with the main blood-stream, that even if the wire could have been passed down so far it could hardly have failed to cause embolisms.

This case, with others that have been treated in a similar manner, shows, I think, conclusively that the method is free from any great amount of risk. But its value as a means of effecting a cure of those aneurisms which usually fall within the province of the physician, as being considered inaccessible to surgical treatment, has still to be estimated.

The following are all the cases of this mode of treatment which I have been able to discover :

(1) Mr. Moore's case, already referred to, where the patient died of acute endo-arteritis and endocarditis with pyæmia.

(2) Dr. Baccelli[1] treated two cases of thoracic aneurism by the introduction into the sac of fine spring-wire. Both cases terminated fatally, but in neither did any ill effects follow the operation; in one, death seems to have been caused by incautious pressure on the sac by the stethoscope in auscultation.

Dr. Baccelli attached great importance to the point whether the communication between the aorta and the sac be small or large, and he appears to have laid down rules for ascertaining this, but as only a brief abstract of his paper is published in the Bulletin these are not given. He considered that the operation was only likely to succeed where the communication was small.

(3). Professor Loreta, of Bologna, in 1885, introduced twenty-two yards of silvered copper wire into a large abdominal aneurism, first making an abdominal section; the aneurism consolidated and became reduced to the size

[1] 'Bulletin de l'Acad. de Méd.,' 1878, p. 18.

of a walnut, and the patient was discharged, apparently cured, on the seventieth day. On the ninety-second day he died suddenly from a rupture just at the junction of the sac and the aorta.[1]

Besides these cases, distal aneurisms have been treated in a similar manner, but these are foreign to the subject of the present communication.

It thus appears that up to the present time no case of aortic aneurism has been cured by this operation, and I am disposed to agree with Dr. Baccelli that it is only where the communication between the aorta and the aneurism is small that this is to be expected. But I think that it affords us the means of consolidating any portion of the sac within reach, and thus we may safely and easily prevent external rupture where this is impending, and may perhaps in some cases relieve pressure on the trachea or other important structures.

[1] 'Memorie della Accad. delle Sci. dell Ist. di Bologna,' vol. vi, 1885.

(For a report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' New Series, vol. ii, p. 59.)

ON THE CHANGES

WHICH OCCUR IN

BONE AND SOFT TISSUES AFTER AMPUTATION OF A LIMB,

AND FROM CERTAIN OTHER CONDITIONS.

BY

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Received December 8th, 1885—Read February 23rd, 1886.

The changes which take place in bone after amputation of a portion of a limb present some interesting features, and are, I have ventured to think, of sufficient importance pathologically, and perhaps to some extent practically, to render the subject worthy of consideration by the Fellows of the Society.

The subject is not a new one. Some of the changes to which attention will be drawn have been remarked on already, but certain other conditions do not appear to have been particularly noticed; and it is this which has led me to hope that a discussion of the subject will not be considered useless or wasteful of time.

The changes referred to are not, however, confined exclusively to bone tissue; to some extent they affect the softer structures.

They are not only found to occur in the bone of a stump of an amputated limb, but also in limbs or parts affected by paralysis. But changes in a marked degree will also be observed, though in a different form and due to a different cause, in bones of parts which have to undergo, or take upon themselves, an extra amount of work, to compensate for the loss of other parts with which they were originally associated and had to act.

My attention was first drawn to this subject by an opportunity afforded me of examining the body of a very old man, who many years previously had undergone amputation of one leg, a short distance above the knee, and had evidently long survived the operation. The subject had been received in the dissecting room of St. George's Hospital; no history could be obtained as to the cause of the amputation, or as to the date of the operation, nor of the subsequent occupation of the individual. Suffice it to say that the stump was well healed and sound, and the cicatrix was evidently of considerable age.

The observations, therefore, as regards this individual case, are simply confined to the description of the more interesting points exemplified in the specimens of bone figured on Plate IX. These consist of the upper portions of two thigh-bones from the same subject, with the head, neck, and great trochanter complete in each. For the illustrations of these specimens I am indebted to my friend Mr. John H. Morgan, Assistant Surgeon to Charing Cross Hospital. The characteristics of the two specimens are accurately represented, and the differences between the bone of the amputated leg and that of the entire femur made very clear.

To indicate accurately the comparative changes illustrated in the drawings, and the specimens themselves, the thigh-bone of the sound side has been sawn through, at a point to make it correspond in length with that of the

amputated side, measured from the upper edge of the great trochanter.

It will be observed, on an examination of the specimens, that the contrast between them is most marked. The general appearance, the thickness, weight, obliquity of neck, and the respective positions of the head of each femur,—all these points tell without trouble which portion of femur must have been taken from the amputated limb, and which belonged to the sound side.

The difference in weight of the two bones is very marked. That taken from the sound side weighed 6 oz. gr. xx. The corresponding portion from the stump weighed 3 oz. ʒiij.

The difference in the obliquity of the neck of the femur of the two sides which occurs after an amputation of a limb through the thigh-bone, is perhaps one of the most interesting and prominent features to be noted, so far as the bone is affected by conditions entirely dependent on, and occurring subsequent to, the loss of a leg above the knee.

It has been found, from the examination of many specimens, that if the subject has lived some few years after an amputation through the thigh, the neck of the mutilated femur will become by degrees very oblique. In the specimen exhibited this is seen to have taken place to a remarkable extent: in contrast to this the neck of the femur of the perfect bone has gradually been brought down to a right angle with the shaft, and lies horizontally between the head and the trochanter. The head of the femur on the side of operation, as compared with the upper edge of the trochanter, is nearly an inch higher than in the opposite limb. The shaft in one is thin and light in weight. The shaft of the other is thickened, hardened, and increased in size beyond its natural growth; more in character with that of a femur of middle age than of one taken from the body of a man eighty years of age.

A most interesting contrast is thus exhibited in these

two specimens. Not only is the neck of the femur of the amputated side seen to be extremely oblique, but that of the sound limb has not only assumed the horizontal position, but the bone itself, neck and shaft, has become thickened, strengthened, and hardened. The extra weight imposed on the sound limb by the amputation of the opposite one, to a great extent, no doubt, assisted to produce this alteration in the neck; the necessary extra muscular action of the sound limb and consequent increased blood-supply was most probably the chief cause of the addition to the substance of the bone. It will thus occur that the increasing obliquity of the neck of the femur on the amputated side gradually adds to the length of the remaining portion of the bone; consequently, for some time after an amputation has been performed, there is a tendency for the stump to become gradually more and more conical, unless precautions have been taken to obviate such an occurrence by the removal of a sufficient portion of the shaft, a fact which should not be lost sight of in the performance of amputation through the thigh.

On the other hand, it will be found that the height of the individual who has undergone amputation through the thigh will diminish to a slight extent, from the circumstance that the neck of the femur on the sound side gradually yields to the extra pressure from above until it has assumed the horizontal position.

The increased obliquity of the neck of the femur after amputation through the thigh is probably due to more than one cause. The removal of the natural weight of the trunk from the head of the bone may exert some influence; but probably more may be due to the fact that the bone is no longer supported from below, but is suspended, as it were, from the cotyloid cavity; it may also be partly owing to the daily decreasing support from the surrounding muscles of the stump. The deterioration observed to take place in the bone after an amputation of the thigh is not, however, limited to that portion of the bone left to form the stump. Similar conditions of marked

diminished nutrition, and consequent wasting of structure, are found to extend their influence to the pelvis of the amputated side. Specimens of this condition have been kindly brought for exhibition by Dr. Humphry, of Cambridge, and Mr. Howard Marsh.

Dr. Humphry was able to secure a specimen of the pelvis with the stump and perfect thigh-bone, from a subject in which he had amputated through the middle of the femur some years previously. In this instance, the evident loss of substance of the pelvis, on the side corresponding to the mutilated femur, can be at once detected; so marked is it, that no one could hesitate, without examination of the thigh-bones, to pronounce on which side the amputation had been performed.

In the specimen exhibited to the meeting by Mr. Howard Marsh, similar conditions to those seen in the preparation from the Cambridge Museum are to be observed. It shows a diminution in size and thinning on the side which corresponds to the amputated thigh. The history of the case is not recorded. The specimen is from St. Bartholomew's Hospital.

In a living subject who has undergone amputation through the femur certain alterations may be detected, such as are borne out by the examination of these parts after death. I had the opportunity of examining a case under the care of Mr. Henry Morris, in Middlesex Hospital, of which the following are the brief particulars:

W. W—, æt. 49, had had his left leg amputated about the junction of the middle with the lower third of the femur, for disease of the knee-joint, when about ten years of age. The following were the measurements of the respective parts:—

On the amputated side, the measurement round the upper part of the thigh was twenty and a half inches, that on the sound side was twenty-two inches. The measurement of the right half of the pelvis, from the median line of the sacrum behind, to the linea alba in front, was fifteen inches. The corresponding measurement of the opposite side was four-

teen inches and a half. From the anterior superior spine of ilium on the sound side to the middle line of symphysis pubis was six inches and an eighth, while that of the opposite side was only five inches and five eighths.

The trochanter of the amputated limb was much less prominent than that of the perfect extremity and could not be very readily distinguished. It was on a lower level than that of the entire femur. In another case in Middlesex Hospital the patient had had his leg amputated for disease of the knee-joint, some four years previously. The measurement from the median line behind to the linea alba in front on the amputated side, was twelve inches and five eighths. That on the sound side was thirteen inches.

I am not aware that attention has been drawn to the occurrence of this alteration in the aspect and conditions of the pelvis, following on amputation of the thigh. Mr. Hilton[1] some years ago drew attention to a somewhat similar alteration of the pelvis following on hip-joint disease in children, though I cannot find that any allusion is made to the changes referred to, which occur after amputation. He writes: "I have ascertained by examination that the os innominatum on the side of the disease does not grow so rapidly, and finally is not so large as its fellow; hence the area of the pelvis is not symmetrical, and thus may interfere with parturition at the full period of gestation. I may add that this pelvic deformity is most conspicuous when the hip disease occurs in, or continues into, the period of early menstruation."

The deformity here described is, however, to be alone attributed to some arrest of growth, whereas that which takes place after an amputation of the thigh, may occur after growth has ceased, and then can alone be the result of absorption of bone tissue, the partial result probably of diminished action of all muscles attached to that portion of the pelvis.

Similar conditions of wasted or wasting bone structure are to be observed under other circumstances, but all bearing

[1] 'Rest and Pain,' 2nd edit., p. 320.

on the same principle, and illustrative of the fact that wherever there is diminished action there is reduced nutrition, and wherever we find extra action there will be found increased growth.

Loss of substance of bone occurs in many conditions of disease; the wasting of the jaws when all teeth have been parted with, as often seen in advanced life; thinning of bone under conditions of infantile paralysis; or that which is attendant on anchylosis of a joint; all these conditions manifestly indicate a diminished blood supply and diminished nutrition followed by a gradual absorption of bony tissue.

To illustrate somewhat practically and more precisely some of the conditions to which the foregoing remarks apply, I cannot do better than quote Mr. Hilton's observations when referring to these changes in a case of disease of the shoulder-joint: "The anchylosis and its remote effects manifested themselves in this way: the humerus and scapula were dwarfed and moved rigidly together, and, in addition to the rigidity of that joint, the clavicle was short, as compared with the other side, and the chest on the left or shoulder-disease side was not so much developed as on the other; hence the left lung and chest-wall were not in true concord as a part of the respiratory apparatus" (loc. cit., p. 319).

Through the kindness of Mr. Henry Morris I am able to exhibit a very interesting specimen of the wasting of bone in association with paralysis. The preparation consists of the bones of a right upper extremity with scapula and clavicle, all showing extreme atrophy; all the bones are very light and fragile. The shaft of the humerus is not thicker than a fibula, and is twisted. The radius and ulna are rounded and about equal in diameter to a goose-quill. Both extremities of the humerus and lower end of the radius have been fractured. The case was no doubt that of an adult, as all the epiphyses are ossified to the shafts of their respective bones.

We not only may observe these changes as taking

place in bones, but the soft tissues, such as muscle, &c., are equally influenced as regards waste or increase under similar circumstances. The waste or increase of muscle may be observed under many conditions.

In the case of a patient the subject of an amputation through the femur we may detect both the one and the other slowly progressing side by side. On the amputated side we find wasting of muscle consequent on diminished muscular action, and lessened blood supply; on the sound side, the substance of the thigh is found to have increased in bulk and the muscles have become largely developed.

A gentleman under the care of Mr. Henry Morris, was the subject of popliteal aneurism of the left leg. The right leg had been amputated by Mr. Nunn eight years previously for ruptured popliteal aneurism. I was requested by Mr. Morris to see the case in consultation with him late one evening. For reasons which need not be entered into it was decided that the femoral artery should be tied without delay. The operation was most successfully performed by Mr. Morris the following morning.

The left thigh had become very stout and muscular, the patient having made constant active use of it, with the aid of crutches and an artificial leg.

When examining the case before the operation, it was found difficult to command the circulation through the aneurism without using considerable pressure over the femoral just below Poupart's ligament. This difficulty was evidently occasioned by the quantity of soft tissue between the skin and the artery, and this accumulation of fat rendered treatment by pressure of the artery out of the question. The amount of pressure necessary to command the circulation would most certainly have shortly produced slough or ulceration of the skin.

The artery was tied in Scarpa's triangle. When the sartorius muscle was drawn to one side its increased size was a very marked object of attention; its breadth being quite twice that of the usual size of this muscle. Similar

enlargement was observed in the deeper muscles, so that the femoral sheath lay deeper than I had ever previously observed it.

Mr. Morris kindly allowed me to take measurements of the respective sides a few days after the performance of the operation. On the sound side the measurement round the thigh close to the groin was 23 inches and $\frac{10}{16}$ths. On the amputated side the measurement round the stump equally near the groin was 21 inches and $\frac{11}{16}$ths, a difference of nearly two inches.

The trochanter on the amputated side was lower than that of the perfect leg, but its outline could not be very clearly defined.

Sir Benjamin Brodie, to whose teaching I owe not a little, drew attention to the wasting of bones when limited in their natural movements. He writes, "You will observe that all bones in a state of inaction lose a great part of their phosphate of lime."[1] I cannot, however, find any allusion to the compensatory growth and thickening that takes place in bones that have a double duty imposed on them.

Sir James Paget also remarks, "We have seen that when a part is, within certain limits, over-exercised, it is over-nourished; so, if a part be used less than is proper, it suffers atrophy."[2]

Mr. Curling drew attention to the changes which occur in bones after injury, but I cannot find that he refers to the alteration of shape, or increase of growth due to pressure or over-exertion.[3]

The contrast between the injured and sound bones is well illustrated in Cheselden's 'Osteographia.' Mr. Curling adds "that bones as well as soft structures fade and waste away when their activity is diminished or their functions suspended. This is seen in the bones of stumps after amputation, and in bones of anchylosed limbs. In

[1] 'Lectures on Pathology and Surgery,' 1846, p. 409.

[2] 'Surgical Pathology,' p. 86, 1863.

[3] 'Med.-Chir. Trans.,' vol. xx, 1837, p. 341.

the new museum adjoining the École Pratique at Paris, founded by Dupuytren, there is a remarkable skeleton of an adult in which all the bones in the body are anchylosed, excepting the lower jaw and the bones of the shoulder-articulations. The bones of the extremities are very much atrophied, the thigh bones being scarcely larger than an ordinary radius."

When we come to estimate the results likely to be observed after the amputation of a limb through the thigh-bone we have to consider (1) the function and the action of the muscles, (2) the weight of the body exerting an extra pressure (on the sound side), and (3) the entire removal of all pressure from the stump.

No longer of much use, no longer pressed upon, no longer exercised in proportion to the opposite limb, the whole stump and corresponding side of the pelvis become affected in a somewhat similar manner. On the sound side the bone has to support more than its natural weight, the muscles have to undertake more than their natural duty; the limb in fact has to perform all, if not more than, the work of two legs; and so bone and muscle are proportionally increased in size, and to some extent altered in shape, while the vascular supply is rendered equal to the demand.

I must again refer to a remark of Mr. Curling's in connection with the rather rapid absorption of bone from non-use. He says in reference to the case quoted from Cheselden, that the late Mr. John Shaw attributed the thinning of the femur to the want of exercise; but adds that "the wasting had taken place to too great an extent, in a short time to be accounted for in this way alone."

I venture to express the opinion that though the wasting of bone under such circumstances as we have considered is necessarily slower than that of muscle, in both it is often more rapid than may be generally supposed. We witness the rapid falling away of muscle in the early stages of hip-joint disease, and had we the power or means to test the waste of bony structure in its commencement,

and early stages of deterioration, my impression is that we should find the process of absorption, simply as the result of inaction, sufficiently active to account for the changes observed in Cheselden's case.

I further venture to express the opinion, after some little observation, that bone tissue and muscular fibre, under certain circumstances, take on more rapid increase than is often suspected.

In an instance in which the first, second, and fourth fingers were removed by me for an accident, the thumb and remaining third finger soon became so mobile that the mutilated hand might be said to be almost as useful as the original one. Within a few months both thumb and finger were decidedly larger, broader, and longer than the corresponding portions of the fellow hand, and in the course of some year and a half a marked increase in size and length in both had taken place.

We are all aware that the slow changes which are constantly going on in bones from birth till death are regulated and modified, so as not to interfere with the form, substance, and strength of their respective parts, so long as healthy action is permitted, and maintained. But as age advances and movement becomes more limited, bone commences to lose its solidity and becomes more oily and is rendered more brittle.

What, however, appears to me to be the most interesting and important question in connection with the specimens figured in Plate IX is the fact that we find in one and the same subject and at the same time two very distinct conditions in the thigh-bones of the opposite limbs; two very distinct and different processes, carried on from the time of the amputation of one limb, to the death of the individual who is certified to have lived over eighty years. On the amputated side the remains of the femur are thinned, oily, and brittle. On the sound side the bone is thick, compact, and firm. In the first, motion, and consequently nutrition, have been interfered with, and we witness the progress of decay. In the second, muscular

action has not only been well preserved, but greatly increased, and here we find the part equal to all the conditions of bone in earlier life.

If such conditions are found to occur under certain known circumstances, is it unreasonable to assume that these facts may, with some slight advantage, be borne in mind in the treatment of certain affections of the osseous system, dependent, not on disease, but on general constitutional disorder? So that, by a careful combination of exercise, position, and rest, combined with the judicious use of mechanical appliances, we may accelerate the improvement of whatever defects such conditions produce.

Subjoined is a short table of specimens illustrative of deterioration of bone, consequent on amputation, paralysis, &c.

LIST OF SPECIMENS ILLUSTRATIVE OF DETERIORATION OF BONE, CONSEQUENT ON AMPUTATION, PARALYSIS, &c.

1. *S. D.*, 51, *St. Thomas's Hospital Museum.*

A right hip-joint, showing complete bony anchylosis; a section has been made through the bones from side to side. Externally the form of the joint is but little, if at all, altered; the margin of the acetabulum may be traced without much difficulty. The cut surfaces show such intricate union that the crusts and cancellous tissues of the bones are continuous, and it is impossible to distinguish their boundaries. The bones are very heavy and their crust is very compact and ivory like. *The pelvis in this case shows evident thinning of ilium in centre.*

2. *S. D.*, 20, *St. Thomas's Hospital Museum.*

An elbow-joint, in which the total destruction of the articular cartilages and partial absorption of the articular end of the humerus had been followed by firm ligamentous anchylosis, more especially between the humerus and ulna. But chronic inflammation, accom-

panied by growth of irregular bony spiculæ from the ends of the bone, and the repeated formation of abscesses, gave rise to constitutional irritation, sufficiently severe to render amputation necessary. *The preparation shows evident wasting of bone from non-use.*

3. *S. C.*, 62, *St. Thomas's Hospital Museum.*

Preparation shows obliquity of neck of femur well marked, after amputation through thigh. No history.

4. *S. C.*, 2, *St. Thomas's Hospital Museum.*

Atrophy of humerus after fracture; upper half of bone remarkably atrophied. Cancellous structure of ununited epiphysis of the head is to a great extent removed, and replaced by soft fat. From this point to the middle of the arm the shaft is exceedingly slender, measuring in the thinnest part a quarter of an inch from before backwards, and rather less from side to side; the long circumference and the medullary cavity appear to retain their proportional size. In the lower half the bone has been fractured in three places. There is, however, no osseous union between the fragments; but they are surrounded on the outer side by an adherent periosteum, and thickened and condensed fibrous tissue, which is also prolonged between their extremities, and unites them more or less perfectly to one another. The same kind of tissue is prolonged into their medullary cavities. *The fragments are much thicker than either the upper or lower portions of the humerus.*

5. *S. C.*, 51, *St. Thomas's Hospital Museum.*

Upper part of femur after amputation. The end of the stump is rounded and for some short distance above this, especially on the posterior aspect, the thickness of the bone is increased by new periosteal deposit. *There is well-marked obliquity of neck.*

6. *S. C.*, 4[1].

Upper part of femur after amputation. The bone gradually tapers towards its lower extremity. *Obliquity of neck well marked.*

7. *S. C.*, 4[2].

Upper part of femur, after amputation, immediately below lesser trochanter. The section that has been made shows well the atrophied condition of the bone, and the closed medullary canal. *Obliquity of the neck of the femur very marked.*

8. *Spec.* 347, *Middlesex Hospital Museum.*

A vertical section of the greater part of a left tibia and fibula, with the tarsus and metatarsus, showing extreme atrophy from disease of leg (paralysis ?). *The compact tissue is reduced to thin shell,* and in places perforated by foramina, due to its total conversion into spongy bone. The greatly expanded medullary cavities, in the recent state, were filled with a pinkish-yellow fatty material from the degenerated medulla. The growth of the bones has been retarded, and the tibia and fibula are markedly curved, the convexity being forwards.

9. *Spec.* 348, *Middlesex Hospital Museum.*

The upper portion of a tibia and fibula from an amputated stump. The bones, especially the fibula, are much atrophied and very light. Their sawn ends are united by bone, and pointed. This was in the case of an adult, as the epiphyses are ossified.

10. *Sp.* 349, *Middlesex Hospital Museum.*

The bones of a right upper extremity, *with scapula and clavicle showing extreme atrophy.* All the bones are very light and fragile. The shaft of the humerus is not thicker than the fibula of a boy, and is twisted. The radius and ulna are rounded, and about equal in diameter to a large goose-quill. Both extremities of the humerus and the lower end of the radius have fractured, possibly in removing or mounting the specimen. An apparent deformity of the hand is probably due to the same cause. This case was no doubt that of an adult, as all the epiphysial points are ossified to the shafts of bone.

Series 1, 2, *St. Bartholomew's Hospital Museum.*

A scapula and part of a humerus. The arm had been amputated long before death, and through disease the bones are atrophied, but the humerus in a much greater degree than the scapula. The shaft of the humerus has less than half its natural diameter and tapers to a slender cone, at the end of which is some rough bone. The marks of the attachments of muscles on it are nearly obliterated, and its texture is high and dry. The head of the humerus is flattened and almost entirely absorbed, and there is a corresponding diminution and change of form in the glenoid cavity.

Series 1, 3, *St. Bartholomew's Hospital Museum.*

Sections of a stump of a humerus, exhibiting the results of atrophy from bony disease after amputation. Its sawn end tapers to a cone; the walls of the shaft are less than a pin in thickness, light and dry; and nearly all the osseous part of its cancellous tissue being removed, the medullary tube appears, after maceration, like a smooth-walled cavity.

S. 1, 4, *St. Bartholomew's Hospital Museum.*

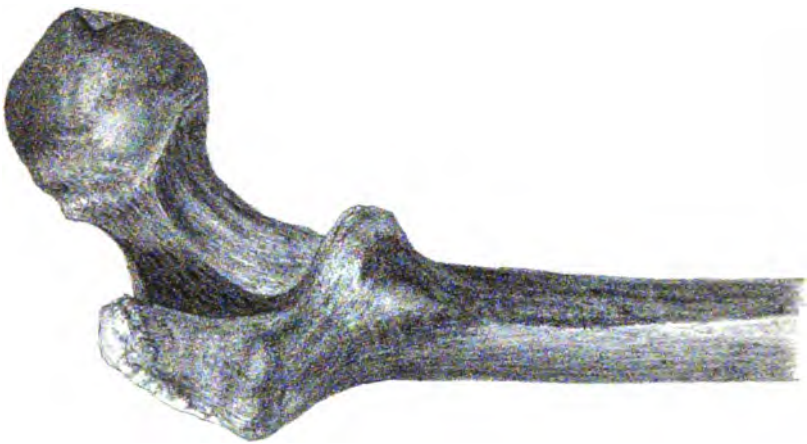
Pelvis and lower extremities of a young man. All the bones of the right side are atrophied. The several prominences on the right os innominatum are less marked, and its iliac fossa is more shallow than the corresponding parts on the left side. The bones of the right thigh and leg are all shorter, less in circumference, softer, and lighter than those of the left limb. From the hip-joint to the ankle there is a difference of nearly two inches in the length of the limbs. In compensation for this difference the left foot is directed almost vertically, so that in the erect position of the body (in imitation of which the bones are arranged) the extremities of both limbs are at the same level. All the bones of the right foot are slender, small, and soft. The arch of the sole is much increased by the posterior part of the os calcis projecting more than usually downwards. The shaft of the left femur is enlarged by external deposit of new bone. The muscles of the right limb were small and in a state of fatty degeneration. The limb had probably been affected by infantile paralysis.

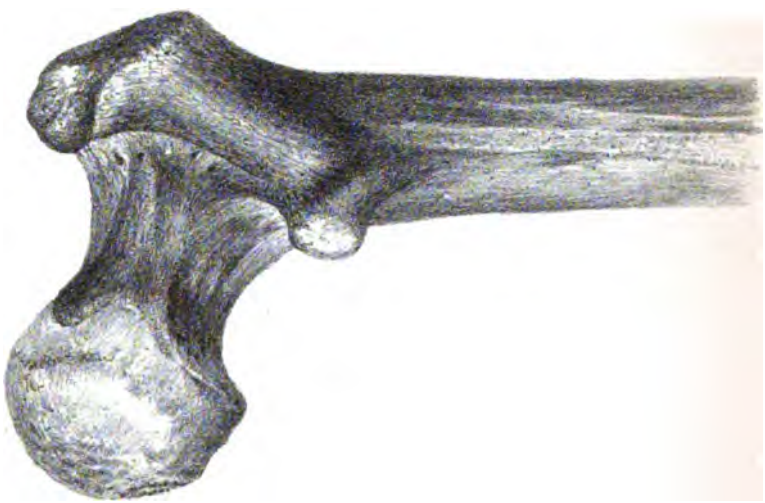
(For a report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' New Series, vol. ii, p. 65.)

DESCRIPTION OF PLATE IX,

On the Changes which occur in Bone and Soft Tissues, after amputation of a limb, and from certain other conditions. By GEORGE POLLOCK, F.R.C.S.

Upper portions of two thigh-bones from the same subject. For full description, see p. 276.





Mintern Bros. lith.

A CASE OF GENERAL SEBORRHŒA

OR

"HARLEQUIN" FŒTUS.

BY

J. BLAND SUTTON, F.R.C.S.

Received December 15th, 1885—Read March 9th, 1886.

THE condition presented by the fœtus, the subject of this paper, although a very rare one, has received a variety of names, *e. g.* :—*Congenital Ichthyosis* (Hebra), *Intra-uterine Ichthyosis, Congenital Hypertrophy of the Epidermis* (Sievruk), *Diffuse Keratoma* (Kyber), *Cutis formatio præternaturalis* (Vrolik). Dr. Wilks refers to it as the "harlequin" fœtus, a term by which it is usually recognised and one worth retaining, but as the name *General Seborrhœa* expresses the nature of the disease, it has been placed at the head of the paper.

The present specimen was sent to me by my former pupil, Mr. Gittings, who is in the habit of furnishing me with fœtuses presenting abnormal conditions. The history of the mother and the circumstances of the pregnancy have no bearing on the case, except to note that she had previously borne several healthy children.

The fœtus was born at full time, and is of the average

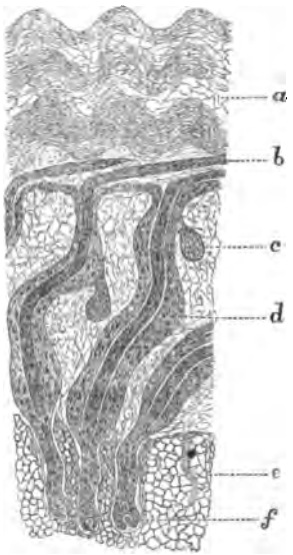
size, weight, and measurement. At a glance, the appropriateness of the term "harlequin" fœtus strikes one (see Plate X). Dr. Wilks[1] describes it thus:—"The impression which is first conveyed to your mind by looking at them is, that the skin had ceased to grow at a certain period, while the tissues within, continuing to increase, had caused distension even to bursting, and thus the integument is cracked and fissured on the most prominent parts of the body." For the most part the fissures maintain a direction transverse to the long axis of the body, but are intersected at right angles by vertical fissures, so that an appearance is produced not unlike that presented by a brick wall. The fissures are most marked on the head, trunk, and trunk end of the limbs. The skin of the hands and feet is free from cracks, but presents a curious cere-like appearance, and the toes are tucked in and seem as though drawn together by the contraction of the skin, giving them a peculiar hide-bound look. The reddish-coloured tissue at the bottom of the fissures is true skin, and if the thickened patches be gently scraped they easily separate from the dermis beneath. The hair on the scalp is matted together by the morbid material, the eyelids are widely open, the tarsal margins are in a condition of lippitudo, and at birth presented a red line, as if of inflammation. The ears are almost obscured by being surrounded with the morbid material. The corneæ, mucous membranes, and viscera, are to all appearances normal.

Microscopical examination of the skin shows that the changes are confined almost exclusively to the epidermis, which in some places, especially on the scalp, exceeds its normal depth about ten times. The superadded tissue is for the most part homogeneous, but in the trunk a laminated arrangement is obvious. On teasing, oily material and epidermal *débris* crowd the field of the microscope.

The thick crust-like masses on the scalp are very instructive when examined in sections, for the examination

[1] 'Pathological Anatomy,' 2nd ed., p. 596.

throws important light on the nature of the disease. The "plaques" on the scalp are, as in other parts of the body, entirely in relation with the epidermis, but instead of the *lanugo* passing directly through the whole thickness of these crusts the individual hairs are coiled and strewn about them in the utmost disorder, exactly as one would expect to find them if a quantity of melted wax were suddenly poured and allowed to set on a hairy scalp.



Section from the skin of the scalp of a harlequin fœtus.
a. Thickened epidermis. *b*. Bent hairs. *c*. Sebaceous glands.
d. Thickened hair sheaths. *e*. Fat. *f*. Papilla.

From a careful consideration of the facts I am convinced that we have in these cases to deal with increased activity of the sebaceous glands, which, about the fourth and fifth months of intra-uterine life, are normally exceptionally active. The secretion of these glands mixed with desquamating epidermis constitutes the well-known "smegma embryonum" or vernix caseosa, which instead

of being shed into the amniotic fluid, cakes or solidifies on the skin and produces the remarkable condition seen in the specimen. Of course it is possible that there is a coincident dermatitis.

That the abnormal thickening of the skin is due to the vernix caseosa receives support from the circumstance that it is most abundant in those parts of the body where this secretion is most copiously formed, viz. the scalp, the ears, on the trunk, especially the flexor aspect, the axilla, flanks, and the neighbourhood of the external genitals.

If this view of the disease be correct, it would be less confusing and more scientific to retain the name "general seborrhœa" to denote the condition, whilst "harlequin fœtus" may be used as an excellent clinical term to serve the purpose of ready recognition.

As the condition is so rare, and our English literature contains no original drawing of the disease, I have been induced to record and figure the present example as well as to append as far as possible a reference to all the recorded cases.

The specimen described in this paper is preserved in the museum of the Royal College of Surgeons.

References.

KYBER.—Eine Untersuchung über das universale diffuse congenitale Keratom der menschlichen Haut. ('Medizinische Jahrbücher,' Wien, 1880, p. 397.)

SIEVRUK.—De congenitâ epidermis hypertrophiâ. (See Kyber, page 408.) (The account refers to two specimens preserved in spirit in the museum of Moscow University.)

LOCHERER.—Aertzlicher Intelligenzblatt, Jahrgang xxiii. Munchen, 1876.

HOUEL et CHAMBARD.—Bull. de la Société Anatomique, 4me sér.; tome iii, 1878, pp. 574, 575. Microscopical and histological examination of a case of congenital ichthyosis.

VROLIK. Tabulæ ad illustrandam embryogenesin hominis et mammalium. Lipsiæ, 1849. On Tab. 92 are drawings

admirably illustrating the naked-eye appearances of a fœtus presenting "cutis formatio præternaturalis."

WILKS and MOXON.—Pathological Anatomy, 2nd edit., 1875, p. 596. Four specimens are preserved in the museum of Guy's Hospital.

There is a specimen preserved in the museum of the London Hospital.

NAYLER.—Treatise on Diseases of the Skin, p. 67. 1874. Refers to the specimens in Guy's Hospital.

JONATHAN HUTCHINSON.—Lectures on Clinical Surgery, vol. i. 1879.

THOMSON.—Practical Treatise on Diseases affecting the Skin, edited by Parkes, 1850. Refers on page 348 to a case observed by Simon.

BATEMAN.—Practical Synopsis of Cutaneous Diseases. 8th ed., 1836.

HEBRA (F.).—Diseases of the Skin (New Syd. Soc. Trans.), 1866, vol. i, p. 111. Refers to cases by STEINHAUSEN, BEHREND, and SCHABEL. In the German edition (Heft 3, Taf. 9, Fig. *c*) there is a figure given under the name *Ichthyosis congenita*.

ZIEMSSEN.—Handbook of Diseases of the Skin. 1885.

KEILLER.—Lond. and Ed. Monthly Med. Jnl., vol. iii, 1843, p. 694.

Dr. HERMANN LEBERT, in his work Über Keratose. Breslau, 1864, p. 94, gives references to cases reported by Richter, 1792, Hinze, 1820, Steinhausen, 1828, Houel, 1853, H. Müller, Okel, 1855, and Souty, 1842.

The following cases have been observed in calves:

GURLT.—Mag. für die gesammte Thierheilkunde. Berlin, 1850.

LIEBREICH.—Two Cases, Diss. Inaug. Halle, 1853.

HARPECK.—Arch. f. Anat. Physiol. und wissens. Med., 1862, p. 393.

I am much indebted to Dr. T. Colcott Fox for several of these references.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' New Series, vol. ii, p. 76.)

DESCRIPTION OF PLATE X.

("Harlequin" Fœtus. By J. Bland Sutton, F.R.C.S.

F. B. Osmond del. Mintern. Bros. Chromo lith.

ON CARDIOGRAPHY,

WITH SPECIAL REFERENCE TO THE

RELATION OF THE TIME OF DURATION OF VENTRICULAR SYSTOLE TO THAT OF DIASTOLIC INTERVAL.

BY

PAUL M. CHAPMAN, M.D.Lond., M.R.C.P.,

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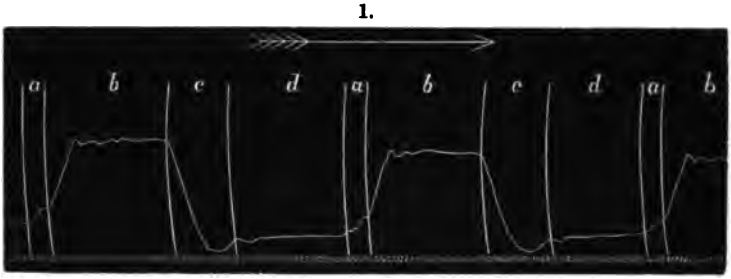
Received November 10th, 1885—Read March 9th, 1886.

The object of the present paper is to bring forward the subject of cardiography, with special reference to the relation of the time of duration of ventricular systole to the time of diastolic interval; to give a short account of some former work in this direction; and to make public so much of the present state of the subject as is due to my own observations. It may also create an interest in the matter which may lead to good results in the future. At the present moment I believe that I am, most unfortunately, the only physician in this country who habitually uses the cardiograph clinically; I myself have only been able to employ it with advantage since I have established a certain basis of comparison to work by. These preliminary experiments and observations having been made, the cardiograph should now come into ordinary use in medicine, and not remain solely an item of the physiological laboratory.

The particular instrument by means of which my observations have been made, and one which is capable of producing very beautiful tracings, is that of Marey as modified by Dr. Burdon-Sanderson; an air-tight tympanum, shaped like a kettledrum in miniature, from the moveable surface of which projects a button which is adjusted to the point of maximum impulse of the heart. The interior of the tympanum is connected by means of a piece of elastic tubing with a second tympanum, to which is attached a lever which marks on a revolving drum tracings of the impulses transmitted from the apex of the heart by means of the cardiograph.

The time occupied by a single revolution of the drum being known, the duration of time occupied in the production of any part of the tracing may of course be measured, by means of ordinates curved according to the length of the lever, which is the radius of the curve. The time occupied in the production of any part of the tracing may be measured quite easily to the 200th part of a second.

1.



a. Auricular systole. *b*. Ventricular systole. *c*. Cessation of ventricular contraction and fall of lever. *d*. Gradual filling of ventricle previous to auricular contraction.

In Fig. 1 a normal tracing is given. I may incidentally mention, in order to show how easily tracings may be taken with practice, that it is an exact copy of one taken by myself from my own heart, without assistance in managing the apparatus. The whole cardiac

revolution occupies ·9230″, of which the ventricular systole occupies ·3260″ and the diastole ·5970″. The pulse-rate is 65 per minute; the auricular systole occupies ·0650″, about one fifth of the ventricular systole.

Experiments were made, to determine the duration of the various parts of the heart revolutions, by Dr. Landois, and published by him ten years ago.[1] Dr. Landois made out an elaborate table of measurements for a single heart revolution at a pulse-rate of 55 per minute. Briefly, his duration of ventricular systole is ·346″, corresponding almost absolutely to my own measurement of ·343″; but he places the duration of auricular systole at ·170″, which, according to my own experiments, is too long, as I have not found it to exceed ·100″, while it is usually less.

Dr. Landois does not attempt to determine the duration of ventricular systole at different pulse frequencies. Any experimenter would soon find that the duration of ventricular systole declines with any increase of frequency of the pulse, and it becomes obvious that, before it would be possible to use the cardiograph for clinical purposes, and to estimate any alterations in disease, it would be necessary to make out by what regular manner, if any, the duration of ventricular systole declines.

It will be within the memory of many that details of experiments were published in the year 1871 by Dr. A. H. Garrod[2] to establish the duration of ventricular systole for different rapidities of pulse. His experiments were made with the earlier instruments of Marey. It is incumbent on me to criticise his results as I have found them to be valueless. The very tracings he published are not in my estimation satisfactory; and the mathematical formula given by him for determining the duration of ventricular systole at any given pulse frequency is not only vexatiously troublesome to use, but is based on

[1] 'Graphische Untersuchungen über den Herzschlag,' Berlin, 1876.

[2] 'Journal of Anat. and Phys.,' vol. v [second series, vol. iv], pp. 17—27.

incorrect observations, and necessarily furnishes incorrect results.

Dr. Garrod's statement, in his own words, is this:—"On comparing traces of different rapidities, it was found that the length of the *first* part varied very definitely, inversely as the rate; not so quickly, but as its square root; and the number of measurements that have been made seems to justify the law that, in health, the length of the first part of the heart's beat varies, for a given position of the subject, inversely as the square root of the rapidity."

Further, in a paper[1] on the "Mutual Relations of the Apex Cardiograph and the Radial Sphygmographic Trace," Dr. Garrod makes the following statement:—"The first cardiac interval is that which occurs between the commencement of the systolic rise and the point of closure of the aortic valve, in cardiograph traces. The number of times that this interval is contained in its component beat is represented by y. The law as to its length may be stated thus: $xy = 20\sqrt{x}$," x representing the frequency of beat per minute.

The calculation of the length of the systole for any given pulse-rate by means of this very cumbrous formula could scarcely be tolerated were the result correct, as it involves several separate calculations. If, when the sum is worked out, we find the result is not in accordance with measurements obtained by experiment, the whole formula may be dismissed with a sense of relief. I should, however, before doing so, justify myself by furnishing some calculations published in Dr. Garrod's paper ('Proceedings of the Royal Society'):

Rapidity of pulse.	Length of 1st cardiac interval.		
	Of minute.		Of second.
36	·0083033	...	·49819
49	·00714286	...	·4285716
64	·00625	...	·375
81	·00$\dot{5}$	...	·3$\dot{3}$3
100	·00$\dot{5}$	...	·300
121	·00$\dot{4}\dot{5}$	...	·27$\dot{2}\dot{7}$

[1] 'Proceedings of the Royal Society,' Feb. 23rd, 1871.

Following his formula, I have calculated out what would be, according to Dr. Garrod, the duration of systole in parts of a second, for every 10 beats increase of frequency per minute from 50 to 130.

Pulse-rate	50	...	Duration of systole ·424″
„	60	...	„ ·384″
„	70	...	„ ·357″
„	80	...	„ ·333″
„	90	...	„ ·317″
„	100	...	„ ·300″
„	110	...	„ ·287″
„	120	...	„ ·273″
„	130	...	„ ·263″

My own table is the result of experiments conducted on upwards of 150 different healthy people, all recumbent. Many of these, again, were caused to vary the pulse-rate by means of exercise, or a bath (the latter leading to various fallacies), or were observed under excitement which quickened the pulse. This table, which has been indispensable to me, and will be so, I hope, to others whom I trust I may attract into this field of investigation, is based upon no theory, but is entirely the result of experiment. Before giving it, I must state, and emphasize the fact, that variations from it are constantly noticed in healthy people, and even in the same person under different conditions, and that these variations may take place within a limit of ·02″ either above or below the measurement given, though I consider this to be the maximum variation in health. I may with confidence and safety state that any variation exceeding this limit may justly be put down as abnormal, and that for high pulse-rates I do not allow a maximum of ·02″ *above* the duration of systole set forth in the table. The maximum is usually obtained with the lower pulse-rates, and I do not allow that for a low pulse-rate it should be less than what I have given in my table.

Table of duration of ventricular systole and of diastole (including auricular systole) of heart, for different rates of pulse.

Pulse-rate.	Systole.	Diastole.	Total.
45 ...	·3600″ ...	·9733″ ...	·1·3333″
50 ...	·3515″ ...	·8485″ ...	1·2000″
55 ...	·3430″ ...	·7479″ ...	1·0909″
60 ...	·3345″ ...	·6655″ ...	1·0000″
65 ...	·3260″ ...	·5970″ ...	·9230″
70 ...	·3175″ ...	·5395″ ...	·8570″
75 ...	·3090″ ...	·4910″ ...	·8000″
80 ...	·3005″ ...	·4495″ ...	·7500″
85 ...	·2920″ ...	·4140″ ...	·7060″
90 ...	·2835″ ...	·3831″ ...	·6666″
95 ...	·2750″ ...	·3566″ ...	·6316″
100 ...	·2665″ ...	·3335″ ...	·6000″
105 ...	·2580″ ...	·3121″ ...	·5701″
110 ...	·2495″ ...	·2959″ ...	·5454″
115 ...	·2410″ ...	·2807″ ...	·5217″
120 ...	·2325″ ...	·2675″ ...	·5000″
125 ...	·2240″ ...	·2560″ ...	·4800″
130 ...	·2155″ ...	·2460″ ...	·4615″
135 ...	·2070″ ...	·2374″ ...	·4444″
140 ...	·1985″ ...	·2301″ ...	·4286″
145 ...	·1900″ ...	·2238″ ...	·4138″
150 ...	·1815″ ...	·2185″ ...	·4000″

The table represents, in decimal parts of a second, the time occupied by systole, or by diastole, of the heart in health for every increase in frequency of 5 beats per minute between 45 and 150. It will be observed that, for every 5 beats increase in frequency per minute, there is a constant decrement in the duration of ventricular systole of ·0085″, my measurement of the duration of ventricular systole at a pulse-rate of 55, viz. ·3430″, almost exactly corresponding with that of Dr. Landois, which was ·3460″.

Though my measurements do not agree with those of Dr. Garrod between 80 and 100, yet I should notice that the decrement between 80 and 100 is the same in both cases.

I must point out certain facts which can be calculated from this table, and which bring to light very forcibly the importance of the diminution of the time of persistence in contraction of the ventricle being a regular and constant quantity. It should be well understood by every physician, that the fact that the time occupied by the ventricular systole diminishes by a constant quantity with increased rapidity of pulse, is one of the greatest importance to the welfare of the economy. By means of the table *the time daily spent in work* by the heart, and *the period of rest* which it enjoys will be for the first time made manifest; *the amount of work done* being to a great extent a separate question, but being also to a great extent connected with the time expended in labour.

By multiplying the duration of systole for one cardiac revolution by the pulse-rate we get the time the ventricle expends in contraction per minute. At 75 the expenditure is 23·175″ in the minute, at 80 it is 24·040″. Thus, for an increased pulse frequency of 5 in the minute, between the pulse-rates of 80 and 85, we find there is an increase in the time expended in contractions per minute of ·865″, or nearly one second.

Now, at a pulse-rate of 120 the duration of ventricular systole is ·2325″; the time expended in ventricular contraction per minute being 27·90″. At a pulse-rate of 125 the duration of ventricular systole is ·2240″, with an expenditure of time in contraction per minute of 28·00″. That is to say, for an increased pulse frequency of 5 in the minute, between 120 and 125, there is an increase in the time expended in contraction per minute of ·1″, or only one tenth of a second.

Thus, owing to the constancy of the decrement in the duration of each systole as the cardiac revolutions increase in frequency per minute, we find that the *total duration of contraction in the minute is increased but very slightly when we pass from one high pulse-rate to another still higher.*

By this provision the whole period of diastole or of

rest in health, is never diminished to less than half of the twenty-four hours. At a pulse-rate of 130, the period of rest is twelve and three quarter hours out of the twenty-four. The period of ventricular labour in health, therefore, never reaches half the day.

I have prepared a table in which the periods of labour and of rest of the ventricles during twenty-four hours are set forth for easy reference. It will be observed that, as the pulse-rate increases, and the need of rest grows more urgent, the period of rest lessens less rapidly; and that, after a pulse-rate of 130 is reached, the period of diastole, or of rest, actually increases.

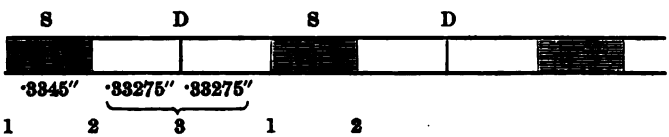
Time occupied in systole or diastole of ventricle during twenty-four hours.

Pulse-rate.		Diastole.		Systole.
45	...	17h12′	...	6h48′
50	...	16h54′	...	7h6′
55	...	16h24′	...	7h36′
60	...	16h0′	...	8h0′
65	...	15h31′	...	8h29′
70	...	15h5′	...	8h55′
75	...	14h43′	...	9h17′
80	...	14h24′	...	9h36′
85	...	14h4′	...	9h56′
90	...	13h47′	...	10h13′
95	...	13h33′	...	10h27′
100	...	13h20′	...	10h40′
105	...	13h7′	...	10h53′
110	...	13h1′	...	10h59′
115	...	12h54′	...	11h6′
120	...	12h50′	...	11h10′
125	...	12h48′	...	11h12′
*130	...	12h47′	...	11h13′
135	...	12h49′	...	11h11′
140	...	12h53′	. .	11h7′
145	...	12h58′	...	11h2′
150	...	13h6′	...	10h54′

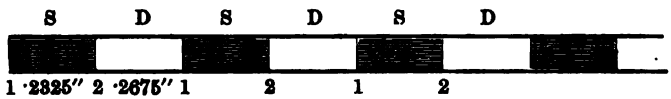
It would be better at this juncture to mention that these facts can be considered in relation with the heart-sounds, and that certain departures from the normal condition may

be roughly estimated by the stethoscope. The first sound of the heart indicates commencement of ventricular systole, the second sound follows immediately after cessation of ventricular contraction, a slight pressure forward of the descending line probably being due to shock of closure of the semilunar valves. Now, in great aberrations from the relative length of systolic and diastolic interval the rhythm of the heart-sounds is different from that in health. Small deviations are of course only made apparent by measurement of a skilfully-taken cardiographic tracing, and could not possibly be detected by the ear.

To consider the healthy rhythm. Where the total cardiac revolution occupies 1·0″ the ventricular systole, or (speaking roughly for the purpose I have in hand) the interval between the first and second sounds of the heart occupies ·3345″, or almost exactly one third of the total cardiac revolution :



On auscultation we can clearly distinguish the rhythm of the sounds in such a normal heart, and could distinctly count "three" in the middle of the pause ; the rhythmical recurrence to "one" falling on the first sound of the next revolution. The sounds of a healthy heart beating rapidly, say at 120, do not take the same rhythm. Normally, for a pulse-rate of 120 the time interval between the first and second sounds is ·2325″, that of diastolic rest is ·2675″, the difference in time in favour of diastole being only 3½ hundredths of a second, which would be inappreciable by the ear. We may therefore in this case assume that the duration of systole and diastole are equal, and that the first and second sounds of the heart would fall thus :



It is to be observed from these facts that in the healthy heart the interval is always less between the first and second sounds than it is between the second and first sounds, even for high pulse-rates; and that therefore auscultation of the healthy heart in no case reveals any departure from the utmost regularity of interval between the sounds, except in the increased interval between the second and first sounds, *i. e.* in diastolic interval, when the pulse rate is low. I have formulated this into a law, stated thus:

In a healthy heart the time interval between the first and second sounds is never less than one third, nor exceeds one half, of the time occupied by an entire cardiac revolution.

In disease obvious discrepancies of rhythm will soon become noticeable to those who make a stethoscopic examination, bearing in mind the law I have enunciated.

To return to my table. I have to indicate the kinds of abnormal cases which show some distinct departure from the measurements there laid down. They are broadly separable into two classes, one in which duration of ventricular systole appears to predominate over diastolic interval, and another in which diastolic interval appears to predominate unduly over duration of ventricular systole. These, again, would each have to be divided, did knowledge permit of it, Class 1 into cases in which the duration of ventricular systole is actually increased, and cases in which the duration of ventricular systole is apparently increased owing to shortening of diastole; Class 2 into cases in which the ventricular systole is actually shortened, and cases in which the shortening is apparent owing to lengthening of diastole.

Abnormalities.

1. I will take first the case in which diastole abnormally predominates over systole. In my experiments on patients who were placed in the dry air (or Turkish) bath, at a

temperature of about 140° F., and sometimes kept there for an hour or more, I found that the duration of ventricular systole occupied less time than it did in the same patient at the same pulse-rate when the tracing was taken under normal conditions. I at first attributed this to the lessened blood-pressure, owing to the dilatation of the capillaries of the skin, thinking *à priori* that if the heart had less obstruction to overcome the systole of the heart would probably be less prolonged. If patients were brought out of the bath and subjected to a cold douche the systole immediately lengthened, with a reduction of the pulse frequency it is true, but regaining the normal duration for the pulse-rate in question.

This I attributed to increased blood-pressure, owing to the contraction of the capillaries and tonic action on the heart by reflex shock.

2.

Faintness in Turkish bath. Systole ·210″. Diastole ·450. Pulse-rate 90.

I induced two young men to submit themselves to simultaneous compression of the large vessels, including the abdominal aorta, but without succeeding in increasing the duration of systole. I also took digitalis for two days, and have subjected a willing patient for three days to the influence of digitalis in large doses, and although I succeeded in decidedly reducing the frequency of contraction I did not increase the duration of systole, allowing for the reduced pulse-rate.

It then occurred to me that possibly the temperature of the blood might reduce the duration of systole, as I had an idea in great simplicity that the contraction of a

muscle in a warm chamber was more sudden and sooner over than is the case when the muscle is in a cooler medium; and, with this view, I took the temperature of the body after long subjection to the bath, and found that I often got a temperature of about 102° F. This again is not to be made much of, since in the case of fevers the systole of the heart is not necessarily shortened in time; and I do not attach much importance to it.

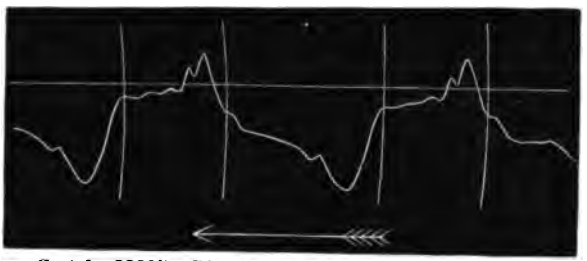
In cases of great exhaustion and prostration I have found the duration of systole very markedly shortened, and my attention was turned to the condition of the patients I had subjected to the Turkish bath. I found that this shortening was most marked in those cases in which the patient was feeling very faint, though it was often unaccompanied with any complaint of faintness. When fainting is imminent, however, it is very marked; and I have found the duration of systole less than normal by more than $\frac{7}{100}$ths of a second (·073″).

It was pointed out to me by Dr. Broadbent, to whom I am indebted for many suggestions and much information, that the cases in which the most marked discrepancy from the normal rhythm of heart-sounds was noticeable by the stethoscope, in the direction of excessive predominance of diastolic over systolic time, were those in which dilatation of the heart was present. Although I do not think this could be demonstrated in every case of dilatation, I certainly have noticed many cases in which, with regular rhythm, the diastolic pause is abnormally long, the systole being short, sudden, and feeble. These cases will improve under treatment, that is to say, as the patient improves in health the rhythm (which is not necessarily *irregular*) approximates more and more to the normal rhythm. For the first suggestion of these facts, as regards dilatation, I am wholly indebted to Dr. Broadbent, who assured me that under iron and strychnine patients would improve in this particular respect, as in others; and, as was to be expected, I have found Dr. Broadbent's observations to be entirely correct. On the

whole I am inclined to think, on consideration of the many cases of comparatively short systole which I have studied, that this condition is not to be attributed to lessened blood-pressure, nor in fever to increased temperature of the blood, but to be immediately due to weakness of the heart muscle and exhausted or defective innervation.

I am strengthened in this conclusion by my observation of the action of nitrite of amyl, the administration of which is attended by dilatation of peripheral vessels and great fall in blood-pressure. The effect on the heart is very well and prettily shown in a tracing taken by myself from my own heart. The height the lever attains is

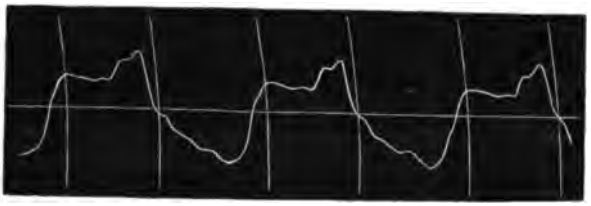
3.



NORMAL. Systole ·3220″. Diastole ·4715″. Pulse-rate 75. Height of initial ascent of lever 8 mm.

reduced, first to 7 mm., then to 3 mm.; the heart is greatly accelerated (from 75 to 116 beats per minute), but it will be observed that the *duration of systole is not*

4.

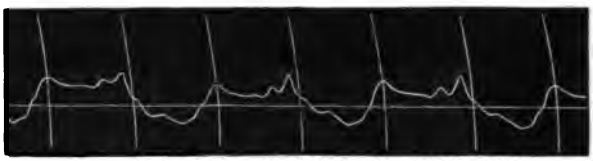


NITRITE OF AMYL (slight effect). S. ·2760″. D. ·3335″. Pulse-rate 98. Height 7 mm.

lessened out of proportion to the increased rapidity of the pulse, but is rather increased in duration.

As I have mentioned the action of nitrite of amyl I ought to say that under its influence the heart tracing sometimes exhibits the phenomenon of dicrotism. There

5.



NITRITE OF AMYL (full effect). S. ·2645″. D. ·2530″. Pulse-rate 116. Height 3 mm.

appears to be a curve or dip during systole in the tracing taken from myself which may possibly mean oncoming dicrotism. I would discuss the question of dicrotism, but the limits of my paper are short and I must confine myself strictly to the matter in hand, viz. the relations between systolic and diastolic interval.

2. To pass to the other class of cases; those in which there is relative excess of systole over diastole. How much this may be due, on the one side, to shortening of diastolic interval, on the other to prolongation of contraction, one cannot say. Using the word fancy to express my lack of scientific proof, I fancy that in most cases it is the shortening of diastolic interval which gives apparent length to the systole. The whole of this subject is of great interest and importance, especially as regards the administration of drugs with a view to their remedial effect. A high pulse-rate need not be immediately dangerous, but let me point out that in these abnormal cases, when systole greatly predominates over diastole, one of the chief things to apprehend is the exhaustion of the patient's cardiac strength. In some cases, in which on auscultation the second sound immediately precedes the first sound (the interval between the first and second sound appearing to be perhaps twice as long as that between the second and first), the heart may be doing forty-eight more

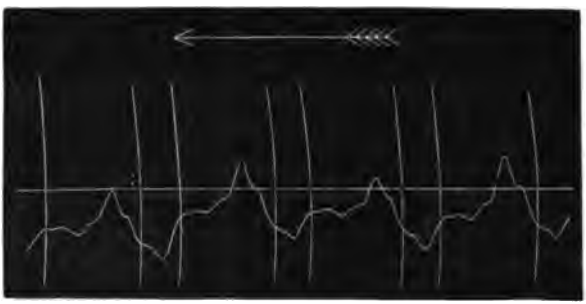
hours' work in the week than it should be doing. In these cases to attempt to slow the heart by prolonging systole might be a grave error. I can give a very interesting, while very short, account of a patient which will bring out these points strongly.

F. J—, a boy æt. 6, was admitted under my care into the Royal Hospital for Women and Children, on March 26th, 1885. Three months previously he had had pains in the knees and ankles, which slightly swelled. He said he was then in bed a fortnight and suffered from sweating. He remained well till a fortnight before admission, since which time he had had pains in the legs and wrists and could not sleep. He looked pale and thin. There was no appreciable swelling of wrists. Temperature 100·8°. The pulse-rate was nearly 150 in the minute. On auscultation a slight systolic murmur was heard at the apex of the heart extending into the axilla.

The sounds of the heart, though rhythmical, did not follow the normal rhythm, which would give an equal interval between both first and second and second and first sounds. The rhythm was altered in such a way that, on listening with the stethoscope, the first sound followed close upon the second sound, the interval between the first and second being about twice as long as that between the second and first.

Two days after, on March 27th, I obtained a tracing from the heart, which I here publish :

6.



F. J—, æt. 6. Systole ·2990″. Diastole ·1035″. Pulse-rate 149.

The time occupied by diastole was so inadequate for rest, and the period of labour was so prolonged in proportion, that, on merely looking at the tracing, I observed to the house surgeon that unless some alteration in the character of the tracing took place the strength of the heart must inevitably fail and the boy would gradually die. I saw him twice afterwards. The state of the heart remained the same. He took digitalis and citrate of potash. Subsequently, on April 1st, complaining of pains in the joints, he took salicylate of soda, which was stopped as he could not retain it. I feared the digitalis harmed him and gave him no more, but tried to support his strength. My treatment was more miserably inefficient than I hope it would be in a future case. The temperature only twice reached 101°, was mostly about 100°, and gradually fell to normal during the 28th, 29th, and 30th, though it rose very slightly during the next few days. There was no albumen in the urine. On my next visit (April 4) the following notes were read to me by the house surgeon:

"Patient began to sink this morning gradually, lasting over many hours. No convulsions, no pain, no insensibility. At 1 p.m. he was almost pulseless and brandy was given. He was very restless for half an hour and said he could not breathe. Was then quiet for a short time, after which he again suffered from dyspnœa. He was again quiet till 2.30, when he again became very restless, and died at 2.40." No P.M. was allowed by the relatives.

Now, I would call attention briefly to the tracing. The period of rest at pulse-rate 149 should be thirteen out of twenty-four hours. The period of rest in my patient was 6 h. 10′. During the week of 168 hours during which he was under my care he had had only forty-three hours' ventricular rest, instead of the ninety-one hours he should have obtained *at the same pulse-rate* had all else been normal. That is to say, his heart had been doing exactly forty-eight hours' more work in the week than it should have done.

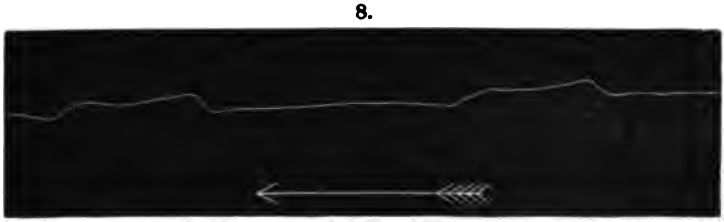
I regret now that I did not largely increase his digitalis to slow the pulse, or administer aconite, the action of which, however, I have not yet worked out.

DIGITALIS.—Digitalis I have since investigated cardiographically, and find, contrary to what I had been led to expect, that it does not lengthen the duration of systole of the ventricles. In the accompanying tracing its action is well seen. The heart was not beating quite regularly before the administration of the drug, the cardiac revolutions are reduced in frequency per minute, the action is regulated, the initial shock seems not to be so great, and there is a gradual rise to the end of systole, which well persists. Thus both systole and diastole are lengthened, the lower pulse-rate itself affording the heart more rest, as can be immediately seen by referring to my second table. Digitalis seems to affect a regulatory nervous apparatus; its salutary effect is best seen in the irregular heart of mitral disease; and I believe it deserves the name of a heart tonic in that respect, and not so much in the

7.

IRREGULAR HEART. Ventricular systole ·3335″ to ·3105″. Diastole ·4600″ to ·7360″ (varying interval ·2760″). Average pulse-rate 67.

8.



EFFECT OF DIGITALIS ON SAME HEART (170 minims of the tincture were taken in forty-eight hours). Ventricular systole (constant) ·3680″. Diastole ·7130″ to ·8740″ (varying interval ·1610″). Pulse-rate 50.

sense of increasing the force and duration of ventricular contraction. I have succeeded by its administration in even making the heart irregular as if by exhaustion of the said regulatory centre.

CONVALLARIA is a heart tonic which probably differs from digitalis in not only slowing the heart, but in actually lengthening the duration of systole of the ventricle. The tracing which I give of the action of

9.

EFFECT OF CONVALLARIA. Ventricular systole ·3795″ (constant). Diastole ·5900″ to ·6555″. Pulse-rate 60 to 63. (Normal systole ·3400″).

convallaria is taken from my own heart, with which I am exceedingly familiar. I took several large doses of the tincture of convallaria in this experiment, thirty to forty minims every half hour for two hours in the morning. I had diarrhœa and a feeling of præcordial constriction in the afternoon, with some giddiness. It will be seen at once, on comparison with my table No. 1, that there is a very perceptible increase in the duration or persistence of ventricular contraction. I have no time to discuss the respective therapeutic advantages of these drugs.

I shall hope to make the action of other drugs on the human heart the subject of future papers.

Besides bringing forward my results I am anxious to popularise the cardiograph. It is an instrument which every physician might have in his consulting room; it is very easy to apply and I have no doubt that a good instrument maker, if he put himself to it, could turn out thoroughly convenient and accurate instruments of this nature at a reasonable price, if there were a demand for

them. Though I do not like to say my table of measurements will require no alterations whatever, I yet believe that it will, for practical purposes, stand any reasonable test. This, the most laborious part of the work, the establishing a basis to work by, has now, to my mind, been done; and I should welcome with great pleasure other workers into a field wherein I feel somewhat solitary.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' New Series, vol. ii, p. 78.)

TWO CASES OF BRONCHIECTASIS

TREATED BY

PARACENTESIS,

WITH REMARKS ON THE MODE OF OPERATION.

BY

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Received November 10th, 1885—Read March 23rd, 1886.

Case 1.—Mr. C—, æt. 67, a gentlemen of literary pursuits and of spare wiry frame, consulted Dr. Theodore Williams February 3rd, 1885. He had contracted bronchitis at the close of 1882, which persisted through the winter and was accompanied by emphysema, and in April, 1883, he had dry pleurisy of the left lung. After this attack the expectoration, hitherto moderate in amount, became profuse, reddish, and purulent, and continued so up to the time of the operation. Patient states that on one occasion he felt something give way in his lung and that he expectorated yellowish fluid for several hours. He spent two months at Malvern and Bournemouth without improvement, the cough increasing, and returned to London in November, and was pronounced by his medical advisers

no better. During the following winter a great variety of medicines and inhalations were tried, but with no permanent benefit, and during the summer and autumn of 1884 the patient seems to have lost faith in remedies and to have discontinued them altogether. He had lost flesh and strength and declared that life was not worth having under the circumstances.

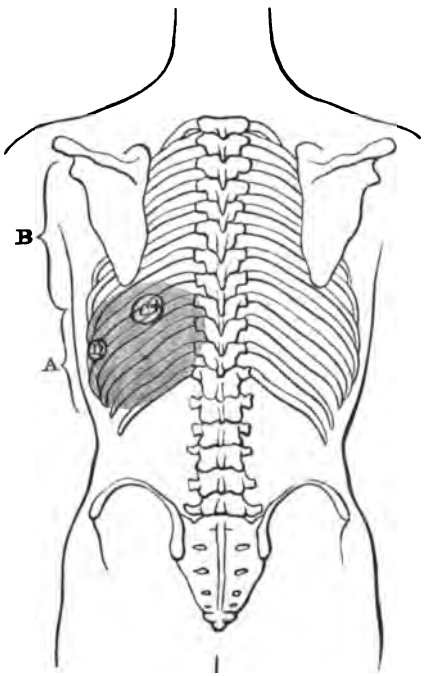
At present his cough is exceedingly troublesome, especially at night, when the paroxysms last for an hour and necessitate getting up and pacing the room. He has an anxious, worn look, and his breath is short on the slightest exertion, the expectoration exceeds one pint a day in amount and consists of frothy pus somewhat sanguinolent and nummular in character. It contains no tubercle bacilli or lung-tissue, but putrefactive bacteria in abundance. Inspection of the chest shows lowering of the left shoulder, with flattening of the anterior left wall, and some deficiency of movement is visible on that side. The heart's impulse is felt in the fourth interspace. The right chest is hyper-resonant and harsh breathing is audible throughout. The left side shows anteriorly considerable flattening, with resonance over the whole surface. Over the lower third of this resonant area vocal vibration is absent and little or no respiration is audible. Over the upper two thirds respiration sounds are harsh.

Posteriorly, dulness commences immediately below the level of the seventh rib behind the mid-axillary line, and following the direction of that rib back to the spine extends then downwards to the base of the lung. The dulness does not vary with change of position, is nowhere strongly marked, and gives the impression of being due to an adherent pleura, and some retraction of the spaces is visible. Vocal vibration is absent over this area, and this absence extends as high as the top of the scapula. Crepitation is occasionally to be heard over the dull area and in one spot (c) about the size of a half crown, situated in the eighth interspace about three inches from the spine, immediately below the scapular angle, some distant tubular

sound can be detected. Harsh breathing is heard over the rest of the lung. Tape measurements at the level of the nipple give the left chest a smaller circumference than the right, by two and a quarter inches.

Two days later Dr. Williams saw the patient in consultation with Dr. Orton, of 30, Lower Phillimore Place, who had had the care of him previously, and a second examination not only confirmed the result of the former one, but also discovered another area of tubular sound, about the same size as the first, situated in the eighth interspace about three inches to the outside of the first (see Fig. 1).

FIG. 1.



A. Area of slight dulness, scattered crepitation and retracted intercostal spaces. B. Area of hyper-resonance and harsh breathing. C and D areas of cavernous sound. + puncture spot.

The diagnosis arrived at was emphysema of both lungs from chronic bronchitis; partial adhesions of the

left pleura, from dry pleurisy, causing contraction of the side, and displacement of the heart's apex; and extensive dilatation of the bronchi in the lower lobe of the left lung.

The adhesion of the pleura over the lower third of the left lung being well ascertained, the next question was as to the number of bronchiectases and their distance from the surface of the lung. From the few and limited areas of tubular sound, and the distant character of that sound, it was concluded that they were limited to the lower lobe of the lung, and were situated at some depth from the surface.

Considering the miserable condition of the patient and the possibility of reaching the bronchiectasis by puncture, the question of an operation in all its bearings was laid before the patient and his wife, and their consent being obtained, Mr. Godlee was requested to perform the operation.

Dr. Williams marked the two areas before described, and directed Mr. Godlee to try the first one, viz. that situated below the angle of the scapula, in the eighth interspace; and to pass a good-sized trocar and cannula to the depth of four or five inches from the skin, directing it forwards and slightly inwards. The patient was anæsthetised by Dr. Orton, and at the innermost of the two spots, viz. in the eighth interspace just below the angle of the scapula, a puncture was made with an exploring trocar about two inches in length, but nothing definite was ascertained. A large aspirator cannula was then inserted to a greater depth, and on making a vacuum, mucus and pus were drawn through it into the bottle. The spray was then turned on, and a T-shaped incision was made through the soft parts, while the cannula was left *in situ*, so that the exact position in the intercostal space which it had occupied might be followed. When this was definitely ascertained the cannula was withdrawn and a scalpel was passed through the intercostal space at the spot. It entered a cavity at a short distance from the ribs, though the exact distance was not

clear; it was certainly not more than, if so much, as an inch. The opening was dilated with dressing forceps, and an attempt was made to introduce the finger, but the ribs were too close together to allow of this being done. A tube was accordingly introduced about four inches long with the usual flange, and through this a considerable quantity (an ounce perhaps), of membranous shreds[1] and pus was forcibly ejected. The tube passed almost directly forwards. Before the patient awoke from the chloroform he began to cough up blood with the expectoration. There was no fœtor about the contents of the cavity; the ordinary gauze dressings were accordingly applied. One grain of opium was administered at night.

February 12th.—Patient has slept well and has scarcely any cough. The expectoration is free from blood, and consists of two or three greyish pellets of mucus. The discharge from the wound has been profuse, soaking through the gauze dressings and reaching the bed. It appears to be thinner and contains a large number of the membranous shreds.

14th.—Yesterday the tube slipped out through the movements of the patient, and, although it has been replaced, the discharge is very slight. The expectoration is of a brick-red colour, somewhat pneumonic in character, with a few streaks of bright hue. Pulse 72, temp. 98·2° F., resp. 20. Crepitation is audible over the lower fourth of the left front chest. A longer tube was then introduced through which the discharge was tolerably free.

25th.—The wound is dressed once in four days, and the dressings found to be quite sweet, though soaked through with thin watery fluid, which scarcely stains them. The tube is extruded by fresh granulations, and has to be shortened half an inch. The sinus has a depth of three and a half inches.

March 2nd.—The patient is up most of the day, and has scarcely any cough or expectoration. The dressings

[1] These were examined microscopically by Dr. Percy Kidd and found to contain no cellular elements, but to consist of amorphous material.

are changed once in five days, and always found to be sweet; the discharge being still watery and soaking through the gauze. The tube has been again shortened. Pulse 72, temp. 98° F.

17th.—The sinus had contracted so much that a shorter tube of smaller diameter had to be inserted, and to-day the increase of granulations has pushed this outside the ribs. Discharge very slight. The tube was removed altogether and the wound dressed antiseptically.

31st.—The patient has gained flesh and looks well. Cough and expectoration absent except on rising in the morning, when two or three pellets of greyish mucus are raised. The wound has healed up.

Physical signs.—The left shoulder is markedly lower than the right, and the movement of the whole side is very deficient. There is curvature of the spine towards the right. Anteriorly the left chest is resonant throughout, and vocal vibration is felt to the very base. Breath-sounds are weak. Posteriorly there is marked flattening, specially from the eighth rib downwards. Here also vocal vibration, formerly absent, is felt to the base, but is not so marked as at the apex or even over the opposite lung. The dulness has disappeared except at the extreme base, and is replaced by marked resonance. Fine dry crackle, chiefly accompanying inspiration, and quite characteristic of emphysema, is heard over the whole posterior surface, but is most marked in the mid-axillary line. No tubular sounds audible anywhere. The right chest remains the same.

May 2nd.—The patient has no fresh symptoms. He has returned to his ordinary habits and drives out on fine days, and also takes walks. He has grown stouter and looks in excellent health. Cough and expectoration *nil.* Measurement of the chest at the mammary level shows the left side to be two and three quarter inches smaller than the right, showing a shrinking of the left side, since the operation, of half an inch. There is more resonance and crackling sound at the left posterior base, showing further development of emphysema.

July, 1886.—Patient remains in excellent health, and walks four miles at a stretch. He has passed through the late inclement winter without fresh symptoms.

Remarks.—The history of this case rendered the existence of bronchiectasis extremely probable, for it may be noted that the expectoration, at first moderate in amount, after the attack of dry adhesive pleurisy became profuse and altered in character, and it is likely that in the lower lobe of the left lung, the wall of the bronchus having been infiltrated during the prolonged bronchitis, had lost its elasticity, and yielding to the inspiratory efforts, and still more to the traction outwards of the adherent pleura, as Hamilton[1] has most ably demonstrated, had become dilated and had lost the power of easily expelling its contents. This explains the limited character of the lesion, and the cessation of the expectoration after the successful tapping of the cavity—the dilatation of the bronchi being limited to the portion of the lung underlying the recent pleurisy. The physical signs indicated bronchiectasis, rather than localised empyema, for the dulness at the left posterior base was nowhere so marked as in effusion, in fact was very slight, and did not vary with change of posture. Moreover, the breath-sounds were not entirely absent anywhere and crepitation of a purely intra-pulmonary character could be heard over the dull area. The intercostal spaces were distinctly retracted. On the other hand vocal vibration was absent, but this was the case not only over the dull spot, but also over nearly two thirds of the posterior surface of the left chest reaching nearly to the top of the scapula, where no dulness existed. The expectoration also had not the character of that of an empyema bursting into the bronchus, which is generally very purulent and rather liquid, whereas this was partly froth and partly nummular pus, streaked with blood, and, for some days after the operation, was distinctly pneumonic. The diagnosis of bronchiectasis was confirmed by the appearance, after the operation, in the discharge from the cavity, of

[1] 'Pathology of Bronchitis,' p. 86.

membranous shreds, which are quite characteristic of bronchial dilatation. In addition to other points of difference, the absence of tubercle bacilli precluded phthisis. The operation appears to have set up some local pneumonia of the neighbouring lobules: as evidenced by the sputum, but had no effect on the patient's temperature, which never rose above 99° F. The entire disappearance of the tubular sounds from the second area after the operation indicated that they originated in the tapped cavity and were conducted through a patch of consolidation to the surface, thus giving rise to sounds similar to those heard over the first area, and as the expectoration practically ceased, we may conclude that only one important bronchiectatic cavity existed and that this was effectually drained. This is no doubt the explanation of the complete success in this case, and although the patient was advanced in years, he had an excellent constitution and was of the lean and wiry kind, which withstands operations well.

Case 2.—Mary E—, æt. 21, single, domestic servant, was sent to the Brompton Hospital for admission under Dr. Theodore Williams by Mr. Hugh Smith, of Farningham, April 1st, 1885.

Her family history was good, with the exception of the death of a paternal aunt from phthisis.

The patient's illness began with typhoid fever eight years ago, followed by cough and expectoration which had persisted ever since; during the last six years the cough had gradually become worse, and the expectoration increased in quantity and fœtor. Seven years ago she had hæmoptysis two or three times, amounting, on one occasion, to a pint, but none since.

The patient had been prevented from taking a situation for some years by the fœtor of her breath.

On admission by Dr. Percy Kidd, in Dr. Williams's absence, she appeared a fairly-nourished but unhealthy-looking young woman. Cough very troublesome. Expec-

toration abundant, 13 to 16 oz., partly frothy and partly muco-purulent and exceedingly fœtid. On examination it contained no tubercle bacilli or lung-tissue. Temperature was 103° F. Pulse 100.

Dr. Kidd's examination of the chest showed on the right side hyper-resonance with some bubbling râles at the base, and on the left side less resonance and bubbling râles throughout. The fœtor was so great that she had to be placed in a private ward.

She improved under treatment, the cough becoming less troublesome, the expectoration diminishing to 4 or 5 oz., and being slightly less fœtid, the temperature falling to 99° F., and the patient gaining in weight; but about the middle of June, in spite of vigorous antiseptic treatment, the expectoration increased and grew more fœtid, and the cough more troublesome and convulsive.

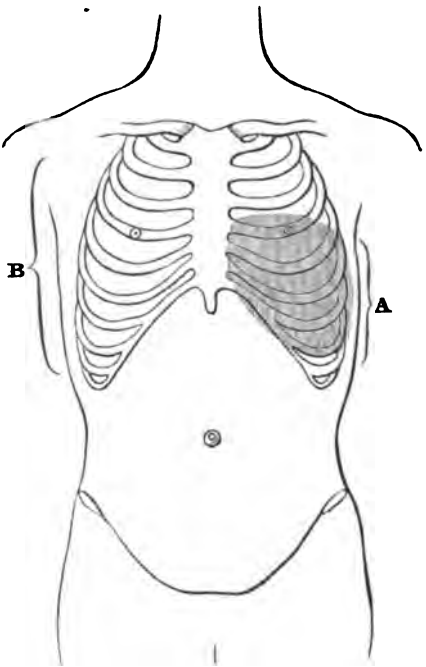
Dr. Williams made several examinations of the chest and noted as follows:—The chest is flattened in the upper left front. The right side is hyper-resonant, with fair breathing except at the posterior base, where a few râles are to be heard. On the left side there is a remarkable absence of vocal vibration; slight dulness and diminished movement extend downwards from the lower border of the third rib in front and from the seventh rib behind the demarcation line, crossing the sixth rib in the axilla, as seen in Diagram 2. The dulness is nowhere strongly marked as in effusions. Above this line there is resonance, and in the axilla, hyper-resonance. The bubbling râles have disappeared from the upper portion of the lung, but coarse crepitation is heard in front from the fourth rib downwards, and behind over about the same area as the dulness. In the sixth and seventh interspaces are two spots (Fig. 3, D and E), each about the size of a half-crown, situated in the axilla, and a third one (F) in the eighth space immediately below the scapular angle. Over these the crepitation is very coarse indeed, especially after coughing. The heart is not displaced. Tape measurements of the two sides give:

	Right.	Left.
At the level of the third rib . .	14½ in. . .	14 in.
At the ensiform level	13½ in. . .	13½ in.

Showing a slight contraction of the upper left chest.

The diagnosis was chronic bronchitis and emphysema of both lungs, followed by pleurisy and fibrosis of the lower lobe of the left lung and consequent dilatation of

FIG. 2.

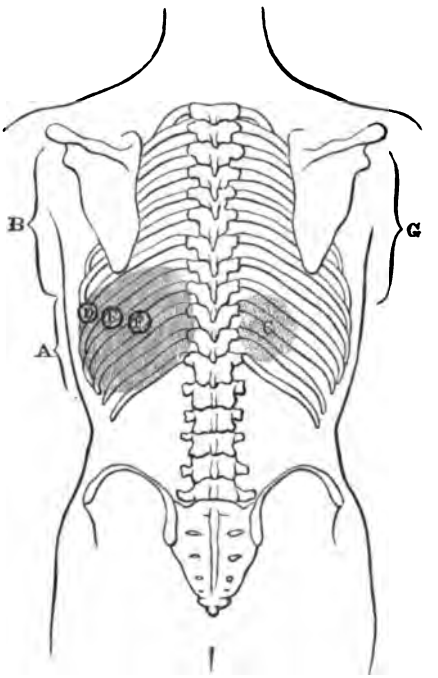


A. Space of dulness, diminished movement, and coarse crepitation.
B. Hyper-resonance.

the bronchi of that side. Dr. Williams was of opinion that several bronchiectases existed, but that three large ones were situated beneath the three areas above described at a considerable distance from the chest surface. He was also of opinion that the dulness was caused by (1) pleuritic adhesion and (2) by fibrosis of the lung. It was

thought that a deep puncture might lay open one of the dilatations and that the other large ones which appeared to lie at no great distance might be connected afterwards and all drained by one tube, although there would be obviously great difficulties in reaching these cavities. The nature and prospects of the operation were duly explained to the patient, who readily consented.

FIG. 3.



A. Shaded space to indicate area of dulness and crepitation. B. Area of marked resonance and harsh breathing. C. Crepitation. D, E, F. Areas of very coarse crepitation. G. Hyper-resonance.

June 29th, 5.30 p.m.—The patient was placed under an anæsthetic, and Mr. Godlee first passed a small exploratory trocar, one inch in length, into the marked spot of the sixth interspace about two and a half inches to the left of the left nipple, and obtaining no result, repeated

the process on the marked spots in the seventh and eighth interspaces with like effect, this operation showing that the pleura contained no fluid. He then inserted a large-sized trocar and cannula of considerable length into the seat of the first puncture (sixth interspace), directing it inwards to the depth of four inches. The trocar was then withdrawn and the cannula connected with an aspirator. Nothing followed at first, but on withdrawing the cannula to a depth of about two inches and exhausting again, some membranous shreds similar to those described in the first case appeared in the receiver. Mr. Godlee now proceeded to open this cavity, but during a paroxysm of the patient's cough the clinical assistant let slip the guiding cannula and the track to the seat of those shreds was lost, and all efforts to regain it failed. Mr. Godlee then with antiseptic precautions made an incision of an inch in length and two inches in depth and passed his finger into the lung, but was unable to discover the cavity. Some free hæmorrhage followed. The wound was washed out with chloride of zinc, and plugged with boracic acid lint soaked in carbolic acid lotion. The antiseptic gauze and pad were then applied.

July 9th.—The discharge not continuing, the tube was withdrawn, and under antiseptic dressing the wound was nearly healed. The expectoration still continues fœtid and cough troublesome. The patient appears to have quite recovered from the operation, eats and sleeps well, and is up and about. Pulse 90, temp. 98·8° F.

Physical signs.—The dulness-area has increased: bronchial breathing is heard over a small spot in the eighth interspace about three inches from the spine, at the same level as the former incision. This spot was carefully marked.

July 16th.—The wound has healed and the patient appears generally fairly well, but the expectoration is unchanged. On examination, the physical signs were confirmed and the area of bronchial breathing again marked. It was decided to make another attempt to reach one or

more bronchiectases, and accordingly the patient was given an anæsthetic, and Mr. Godlee passed a fine curved trocar and cannula into the marked spot to the depth of at first three inches and then of five inches, the curve being directed towards the median line, so as, if possible, to intercept some dilatation of the bronchial tree. Nothing followed but blood. The same trocar and cannula were passed into the old wound and directed first upwards and then inwards, but with no result. Mr. Godlee then, under carbolic spray, made an incision and laid bare the eighth rib, and excised about an inch of it in order to approach nearer to the bronchi before again attempting to puncture. The pleura was carefully examined and found adherent. The trocar and cannula were passed to the depth of five inches. On withdrawing the trocar, a few drops of pus oozed from the cannula. Mr. Godlee then cut down along the cannula and introduced a drainage-tube. No more pus followed at the time, but a good deal of blood. The patient, who had been expectorating the usual fœtid pus, suddenly began to spit clots of blood, evidently coming from the wound. The tube was fixed in and the wound dressed antiseptically.

July 18th.—The wound has been dressed under carbolic spray; the discharge from the tube is distinctly purulent, and has soaked through several layers of the gauze.

July 20th.—The patient is doing fairly. Before the dressings were removed to-day the characteristic odour of the expectoration was noticed to come from them, and was still more marked when they were undone. Discharge profuse and fœtid; wound granulating and healthy.

July 23rd.—Discharge less fœtid to-day. At 5·45 p.m. patient had hæmoptysis, ten ounces, and appeared rather excited. Pulse fairly good. Temp. 98° F.

The hæmoptysis continued for three days, the patient bringing up nine and eight ounces of blood on the second and third days respectively. The blood was bright coloured and had no fœtor. The temperature rose to 103° F., but has fallen to-day to 100° F. The sputum is now blood

stained and eight ounces in amount. Pulse 92, fair. The discharge is scanty and not fœtid, wound healthy.

August 5th.—The hæmoptysis recurred to the amount of three ounces on July 29th, and to a less amount on the 31st, and lastly, there was a small quantity on August 4th. On the last two occasions the amount was small, but on one it was accompanied by slight lividity of the face, cold and moist extremities, a rapid and compressible pulse, and some mental excitement. These symptoms, however, all subsided.

27th.—The patient has improved greatly in appearance, and has gained four pounds in weight, though she lost several during the hæmoptysis. The wound has healed up. Cough is much less troublesome. The expectoration varied from four to six ounces for some days, but has now fallen to two ounces, and is sometimes fœtid and sometimes quite free from odour. The temperature is 98·2° F. Pulse 74, good. The patient sleeps soundly and has an excellent appetite, and declares she feels quite well.

The following chest measurements were taken:

	Right.	Left.
At the level of the third rib . .	14½ in.	14¾ in.
At the ensiform level	13½ in.	13 in.

These show some increase at the upper level, but a diminution at the lower one, in the region of the operations. There is marked flattening and contraction in the neighbourhood of the cicatrices on the left side, and more dulness at the base posteriorly. Fine crepitation is audible in parts, but no bronchial sound anywhere in the dull area. Some tubular sound is heard in the interscapular region, over a space the size of a half crown. Over the anterior surface there is fair resonance, the breathing is much freer, and the moist sounds have entirely disappeared.

Remarks.—In this case we had to deal with disease of apparently long standing, and of constitutional origin, for the symptoms of bronchiectasis followed closely after typhoid fever, eight years previously, and the patient

showed signs of marked cachexia. The probability of bronchiectases in the left lung was easily recognised from the fœtor of the expectoration, and the amount of chronic pneumonia present at the base. The slight dulness and flattening enabled us to trace the line of adherent pleura. But to determine the number of dilatations and their exact position was most difficult, partly from the amount of congestion at the base of the lung, and partly from the presence of emphysema, which existed in the upper parts of the lung. In both operations Mr. Godlee had to puncture in several directions, and in the second, to incise the lung freely before the bronchiectasis was reached.

Though this patient was sent up to the hospital with the view of operative interference, by Mr. Hugh Smith, who had seen one of Dr. Williams's former cases, she was kept in the hospital for two months, and treated with antiseptics, and it was only when these remedies entirely failed and her symptoms became worse, that operation was resorted to. The extensive hæmorrhage which followed the second operation appeared to be due to some ulcerative process set up by the presence of the tube, and not to the operation itself. This led to an increased infiltration at the base of the left lung. The diminution in the amount of sputum and the fact of its being only intermittently fœtid, combined with the general improvement of the patient, are the results claimed for the operation, but the presence of bronchiestases in other parts of that lung, and possibly in the right lung also, precluded complete success.

However, where both cough and expectoration are reduced and the patient's life is rendered fairly tolerable, we may claim a moderate success.

Remarks by Dr. WILLIAMS.—The subject of paracentesis of cavities has been of late years brought before this Society by Dr. Douglas Powell and Mr. Lyell (vol. lxiii), and by Dr. Cecil Biss (vol. lxvii), and also by Dr. Cayley and Mr. Pearce Gould, in the latter volume. As Dr.

Powell's and Dr. Biss's papers contain an account of the principal literature on the subject, I need not enter upon it, but will refer the Fellows of the Society to their papers, confining myself to a short account of my own experience.

Dr. Cayley's case in vol. lxvii, and his case in vol. xii of the 'Clinical Transactions,' were instances of gangrene of the lung treated by paracentesis with relief, and on one occasion with recovery.

Dr. Powell's and Dr. Biss's cases, as well as those two just related, are examples of bronchiectasis, and it is in reference to operative interference in this class of patients that I would direct attention, as, having had in all six cases of bronchiectasis treated by paracentesis, the subject is one of considerable interest to me.

Two of these cases have just been related, and three of the other four have been published elsewhere.[1]

In all these three the cough was convulsive and harassing, and the expectoration so fœtid as to necessitate the isolation of the patient. Various kinds of antiseptic treatment, both general and local, were persistently tried, and failed, before the operation was had recourse to.

The sixth case, hitherto unpublished, was that of a boy, aged thirteen, who had a history of chronic cough and of fœtid expectoration of three years' standing, accompanied on one occasion by hæmoptysis. A good deal of emphysema was present, and signs of bronchiectasis were detected in the left side, especially in the first and second interspaces in front, and posteriorly over the eighth and ninth spaces below the scapula. After the patient had been in the hospital several months, without improving under various kinds of treatment, at my request Dr. Hicks made a vertical incision two and a half inches in length, involving the eighth and ninth intercostal spaces below the scapula, where the gurgling sounds were loudest, and punctured

[1] 'Clinical Transactions,' vol. xii, p. 47; "Lectures on Bronchiectasis," 'Brit. Med. Journ.,' vol. i, 1881, p. 837; 'Proceedings of Medical Society,' vol. vi, p. 323; and 'Lancet,' vol. ii, 1882.

first the eighth space and then the ninth. The second puncture was successful in reaching the excavation, and a discharge was established through a tube. This continued for some time but did not reduce either the fœtor or the amount of the expectoration, and after a while the discharge ceased. Another attempt was then made to reach the principal cavity from the same wound by deepening the puncture. At the depth of four and a half inches the bronchiectasis was reached and a drainage-tube introduced. Scarcely any matter flowed at the time, but the following day the dressings of the wound were soaked through and through with it; air could be heard whistling in and out, and on coughing some very tenacious fœtid muco-pus escaped from the tube. The discharge continued for about three weeks to the amount of two or three ounces a day, but the expectoration did not greatly diminish in quantity though it was less fœtid. At the end of this time profuse hæmorrhage occurred from the wound, apparently due to ulceration from the pressure of the right tube. This was stopped, but it recurred on the introduction of a flexible tube, which it was found necessary to remove altogether, and the wound was allowed to heal up. Later on the patient was attacked with pyæmic periostitis of the left forearm, which was incised, and a good recovery followed. Under these circumstances no attempt was made to reach the other bronchiectases whose position had been detected. Considerable contraction of the punctured side followed the operations and the patient left the hospital, his cough less troublesome and the expectoration less fœtid. He was ascertained to be alive two years afterwards.

In three out of these six cases the diagnosis of bronchiectasis was confirmed by post-mortem examinations, and there is every probability that it was correct in the other three; the general aspect of the patients and the symptoms of the disease amply confirming the physical signs.

The post-mortem examinations in the three fatal cases indicate clearly the mode of death in bronchiectasis. It

seems to be from some form of septicæmia. In two of my cases, and in Dr. Powell's, it was from septic pneumonia of the healthy lung through inhalation of fœtid secretion. As a proof of this, in each of my cases I was able to trace the membranous shreds from the dilated bronchi into the smaller bronchi of the pneumonic lung. Sometimes the septic material enters the stomach and intestines and gives rise to diarrhœa, and sometimes it passes into the circulation, causing pyæmic abscesses. Abscess of the brain was the cause of death in one of my cases and in Dr. Biss's. Another of my patients suffered from pyæmic periostitis. The danger of septic pneumonia from reinhalation is greater than is generally supposed, though undoubtedly many cases of bronchiectasis go on for years without its occurrence; yet I have rarely witnessed this immunity where the expectoration is very fœtid. This point ought to be borne in mind in regulating the posture, and especially the decubitus, of these patients. The mode of death is certainly one strong argument in favour of the operation. Another, which has been advanced by Dr. Powell, is that much of the secretion and the efforts made to expel it from the bronchi are due to the great irritation which the passage of the fœtid matter causes to the bronchial membrane, and this was well shown in Mr. C—'s case, where the whole expectoration diminished from a pint a day to a few pellets immediately on the establishment of the external discharge.

A third argument in favour of this operation is the comparative invulnerability of the lung-tissue, for it has been repeatedly demonstrated that the lung may be punctured to a considerable depth without giving rise to any serious symptoms or marked physical signs. At one of the autopsies we endeavoured to trace a puncture made in the eighth interspace below the scapula to a considerable depth, a few days before death, and entirely failed, the lung having apparently completely recovered. Such recuperative power does the lung display when irritated.

In most of these cases the lung-tissue had undergone fibrosis, and puncturing this tissue seems to promote its growth and subsequent contraction.

One accident that these operations are liable to is inoculation of the pleura with septic material, leading to pleuritic effusion or empyema. This happened in one of my cases, on the withdrawal of the aspirator needle (though a fine one) from the bronchiectasis. Unfortunately the pleura which was adherent over the greater part of the lung was not adherent over the region of the cavity, and hence the accident. As a rule, however, this can be ascertained by observing the state of the intercostal spaces on deep breath, and of course no operation should be attempted if there is any doubt about adhesion.

One accident of these operations is hæmorrhage, which occurred in two of my cases, and was rather troublesome in Case 2 (Mary E—). I allude not only to the hæmorrhage accompanying the operation, but that which followed. It is quite possible that the pressure of the tube against the fresh granulations during the various movements of the patient gave rise to ulceration. In such cases the indication is at once to withdraw the tube.

It will be noted that in all six cases the diagnosis of the cavity was sufficiently accurate to enable us to reach it, and the difficulty consisted less in localising its situation than in ascertaining its distance from the chest wall. In more than one case it was found necessary to deepen the puncture considerably, before it was successful.

The two principal difficulties in the diagnosis of a bronchiectasis cavity, are (1) The presence of emphysema, which invariably accompanies the globular form of bronchial dilatation and often entirely masks the physical signs of a cavity, even when the patient's sensations and the amount and character of the expectoration point to the presence of a bronchiectasis in a certain portion of the lung.

(2) The character of the cavernous sound heard over bronchial dilatations. This is so jarring in tone that it is

audible over a far larger area of chest wall than that immediately overlying the cavity; and it is not rare on this account that the size of the bronchiectasis is thought to be larger than it eventually proves. This jarring note is more common in bronchiectasis associated with interstitial pneumonia and fibrosis and assuming the cylindrical form, than in the globular bronchiectasis accompanied with chronic bronchitis and emphysema.

The success of the operation seems mainly to depend upon whether the bronchiectasis is single, or at any rate confined to one lobe of a lung the pleura of which is adherent, or whether it is multiple, and affects the bronchi of both lungs. In the former case operation gives the greatest possible relief, and may, as in Case 1, effect a cure. In the latter case, and especially where there are indications that the whole bronchial tree is more or less dilated, no advantage can be looked for and the operation cannot be advised.

To sum up, paracentesis in bronchiectasis seems to me to be indicated under the following circumstances:

1. In cases where antiseptic treatment of all kinds, having failed to correct the fœtor of expectoration and to allay the harassing nature of the cough, death by septic pneumonia seems imminent.

2. Where all evidence goes to prove that the bronchiectases are confined to one lung, are situated in the lower lobe, and have overlying them an adherent pleura.

It is not indicated where multiple bronchiectases exist in both lungs, where they are surrounded by emphysema, and where the pleura is non-adherent.

Remarks by Mr. Godlee.—The surgical aspect of the first case presented no difficulty whatever from beginning to end; the cavity being single and the position accurately localised by Dr. Williams, and verified unmistakeably by the preliminary puncture with the aspirator needle; there was a clear indication for cutting down with this as a guide and making a free opening. The pleura was ad-

herent, and the cavity at a short distance from the surface of the lung, so that here again all was straightforward, and in the further progress of the case the sudden diminution in the amount of expectoration and the rapid closure of the cavity, as shown by the decrease of the discharge in the dressing, left no doubt as to the advisability of shortening and ultimately withdrawing the tube.

The second case, however, illustrates most of the difficulties that the surgeon is likely to meet with in dealing with cases of bronchiectasis.

First, the lung containing possibly the ramifications of one or more branched or labyrinthine cavities and the intervening pulmonary tissue being consolidated, the physician is not always able with certainty to define the exact limits of the cavity which it is desired to open, though he may indicate its position generally. The preliminary punctures are thus frequently most unsatisfactory, for it must be remembered that while the individual branches of the cavity may be comparatively small, the bronchi themselves contain a material of a precisely similar nature to that in the bronchiectasis; confusion may therefore easily arise either from just missing an elongated cavity which gives very obvious physical signs, or puncturing and subsequently cutting down upon a bronchus, because some pus has been drawn out of it into the aspirator.

Again, some timidity is naturally felt in introducing a large aspirator needle to a great depth into the lung. It must not be forgotten that the nearer the root of the lung is approached the larger the pulmonary vessels become, and that a puncture through a branch of the pulmonary artery of some size into a bronchus might conceivably lead to very serious symptoms.

Secondly, in this case the pleura was closed by adhesions, but they were so soft that they easily broke down with the finger, so that the lung could be pushed away to some extent from the ribs. If the two layers of the pleura be not adherent two difficulties present themselves (and it must be remembered that however great the *pro-*

bability may be that adhesion has taken place we can never be absolutely *certain* of it until the incision has been made through the chest wall); in the first place, it is impossible to puncture the lung with any amount of accuracy or definiteness, because it recedes before even the sharp point of the needle; and in the second place, if we do succeed in laying open the suspected cavity we run the risk of setting up a septic pleurisy. It is not likely that the lung will be in a condition to collapse very much, for it is probably in a more or less solid state, and it is not likely that the pleura will be found quite free from adhesions, so that a general pleurisy need not be anticipated; still, if the condition mentioned be found, it is safer to stitch the surface of the lung to the opening in the parietal pleura. This is not, however, a very satisfactory proceeding. I have done it on one occasion where the suspected adhesions were not found, but though I succeeded in bringing the lung out to the chest wall, the two surfaces of the pleura did not unite very well, and after a few days it was possible to pass a probe freely into what remained of the pleural cavity, some of the stitches having no doubt cut out through the friable lung-tissue.

Thirdly, there may arise difficulties in connection with the hæmorrhage. The incision of the lung-substance, solidified as it is in these cases, does not in my experience often lead to much bleeding; but it is impossible to avoid the risk of opening a large vessel, at a depth from the surface which would render the application of a ligature out of the question, and it seems to me highly probable that a case will some day arise in which very serious hæmorrhage will occur. In Case 2 the bleeding was very free, but there was no difficulty in controlling it by plugging the wound. This is clearly the only line of treatment to be adopted, and as far as I have yet seen it always (as in the free hæmorrhage which follows incision of the parenchyma of the liver) proves to be successful. But hæmorrhage may also take place into a bronchus and then

cause considerable hæmoptysis. I have now seen this occur three times from the puncture of an aspirator needle, which has no doubt passed either completely through a bronchus into a neighbouring vessel, or through a vessel into a bronchus. This accident it seems impossible to guard against, but it suggests the inadvisability of producing profound insensibility with the anæsthetic, so that the patient may by coughing be able to get rid of the blood from the air passages. In all these cases the hæmorrhage quickly stopped, but in estimating the risks of the operation it must be remembered that M. E— had severe hæmoptysis on several occasions, at a subsequent period in the case; though we must not forget the history of large hæmoptysis before admission.

Another very real danger of which I have seen a striking example is that during the coughing which occurs whilst the anæsthetic is being administered, the bronchi may become dangerously obstructed by the pus which previously filled the cavities. This not only renders the diagnosis of the position of the cavity for which search is being made extremely difficult, but produces more or less asphyxia, which, when added to the narcotic effect of the anæsthetic, may sensibly imperil the safety of the patient.

The indication is to give the anæsthetic slowly so that coughing may be avoided, and in order to prevent the pus from finding its way from the affected to the sound lung, to keep the patient as much on the back as possible.

It is perhaps presumptuous to attempt any general conclusions on the advisability of surgical interference without more extended experience, but the following opinions may be hazarded provisionally.

First, when the physical signs point clearly to the existence of a cavity on one side only and to its being moderately localised, the indication is to operate. But it is wise to make quite sure by preliminary puncture of its exact position before an incision through the chest wall is made, even if this involve exploration on more than one

occasion. Secondly, if the physical signs indicate very extensive mischief on one side only, though the prospect is much less promising, some good may result from operation. If in such a case the preliminary puncture has apparently revealed the presence of a cavity which the further exploration fails to open, the patient will probably suffer no serious consequences from the operation. It is even possible that the incision into the lung and the interference with the natural rigidity of the chest wall may aid in the contraction of the cavity in the neighbourhood of the puncture; while it is just possible that the cavity may have been only just missed, and that by a process of ulceration it may at a later period discharge itself into the actual opening. Thirdly, if the physical signs point with anything approaching to probability to the presence of bronchiectases in both lungs it is wisest to abandon all thought of surgical interference.

(For report of the discussion on this paper see 'Proceedings of the Royal Medical and Chirurgical Society,' New Series, vol. ii, p. 85).

ON SUPRA-PUBIC LITHOTOMY.

BY

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THE high or supra-pubic operation for stone in the bladder has had a singular history. Its first performance may perhaps date from the second century, but the earliest reliable case is that of Pierre Franco who, in 1561, thus succeeded, after the failure of some other method (probably "on the grip") in extracting a large stone from the bladder of a child. The patient recovered easily. Franco, however, ends his account of the case by saying, "I do not advise any man to do the like."[1]

Nevertheless in 1590 Rosset published a remarkable, and for the period a very advanced essay on this subject, which was unjustly blamed and criticised by Hildanus in 1682. Other writers, notably John Douglas,[2] described this operation, but it obtained no repute until Cheselden took it up in 1722, during which year he cut "nine patients this way." They were of various ages, from four to nineteen years. Two died, one of renal calculus,

[1] Pierre Franco, 'Traité des hernies,' p. 139, Lyons, 1561.

[2] 'Lithotomia Douglassiana,' 1723.

there being renal calculus and abscess, the other of some fever, either fortuitous or pyæmic. But the histories record one after another the ease and comfort of the patient, together with the facility of recovery. Yet Cheselden, who about this time was emulous of the success obtained by an imitator of Frère Jacques (Rau, of Amsterdam) abandoned the high for a perineal operation, and soon after perfected the manner now known as "lateral." Since that time the supra-pubic method has only been resorted to when the stone has been diagnosed as very large. It may, however, well be doubted whether surgeons are right in regarding the high operation as one only to be used in exceptional cases, and indeed since 1851, when Günther published his well-known work,[1] there seems to be some disposition to reconsider the question.

My thoughts were more especially led in this direction by a rapid sequence in my clinic of cases of vesico-vaginal fistula. I had under my care in seven months (the latter part of 1883 and beginning of 1884) three cases of this affection, all originating in the extraction of calculi during infancy and youth by different surgeons. Such fistulæ are very recalcitrant to treatment by operation, for they lie in the midst of hard, thick cicatricial tissue. The bladder is much diminished in capacity. In two of the cases the fistulæ were very high, and in the thickened state of parts the uterus could not be drawn down.[2] I do not know how or why these cases should have all come under my notice in such rapid succession, but they made a great impression on my mind, and I determined that when any female child came under my care with a stone too large and

[1] 'Der hohe Steinschnitt,' Leipzig, 1851.

[2] One of these women, aged nineteen, I succeeded after two operations in curing; another, aged twenty-four, had been thrice subjected to operation before I saw her. I gave a guarded prognosis concerning the result of any fresh attempt and have not seen her since. One is incurable, the fistula lying close to the os uteri, which, in the almost cartilaginous hardness of parts, cannot be brought down. She is approaching the menopause; when that occurs I shall occlude the vagina.

hard to be extracted per urethram either whole or in fragments, it should be taken out above the pubes.

CASE 1.—Rose A—, æt. 9, came under my care on 6th February, 1885.

No history was obtainable. The parents simply left the child and did not come again.

She was greatly emaciated and exceedingly fretful. During both night and day she had to micturate very frequently, suffering greatly in doing so. A good deal of urine came away involuntarily, or at all events not restrained. The urine was alkaline, sp. gr. 1019, pale and opalescent from slight admixture of mucus. A little albuminous cloud appeared on boiling. There was a deposit (quarter of glass) which consisted in part of amorphous powder, but very largely of oxalate of lime, chiefly in octahedral crystals.

When a sound was passed into the bladder it immediately impinged on a large stone; if the instrument was pressed further in the same direction a little urine flowed, as though the calculus acted as a valve over the urethral exit; but by a little manœuvring the instrument could be made to pass behind the stone. Examination per rectum revealed little, a good thickness of soft structures intervening between the finger and the stone. Vaginal examination showed that the calculus occupied nearly all the breadth of the pubes and reached a good way upwards.

February 12th.—The child was placed under the influence of ether. A further examination caused me to conclude that the stone was even larger than it at first seemed. About 3½ oz. of carbolized water was injected, when percussion gave dull notes to rather less than half way to the umbilicus. Requesting my assistant to place a finger in the vagina to steady the stone forwards, and at the same time to compress the urethra against the pubes to prevent any outflow of urine, I made an incision about three inches long strictly in the middle line, and after twisting two small vessels, divided the linea alba, taking

care to cut from above downwards. The recti and pyramidales were held apart by broad retractors; but the peritoneum was not in view.

The peculiarly soft yielding tissue which lies between the bladder and the abdominal wall was now divided, and the bladder punctured; when a little water had oozed from the bladder it was opened in a downward direction to the extent of about two inches. The stone thus laid bare was gripped in a small pair of straight lithotomy forceps. They slipped twice, but on the third attempt the calculus was brought about half way out of the bladder. The edges of the recti, however, held it, and the linea alba was therefore divided a little further in an upward direction with a probe-pointed bistoury, superficial to the peritoneum, and the stone was easily extracted. It weighs 2¼ oz. minus 5 gr.; that deficit would be more than outbalanced by the chipping from its upper end. Its length is 2¼, one short diameter 1½, the other short diameter 1¼ inches.

As the bladder contracted and emptied itself the anterior fold of peritoneum slowly descended and came into view.

The cavity examined by the finger was found free from any fragments or detritus; the incision was closed with four catgut sutures. Three quill sutures were passed through the whole thickness of the abdominal wall, bringing together the upper five sixths of the wound; a good-sized drainage-tube was placed behind the recti and protruded at the lower corner of the incision, the skin was sewn with wire, and a catheter passed per urethram.

13th.—Passed a very good night. Temp. 99,° pulse 100. The dressings were found sopped with urine; none passed by the catheter. The lowest superficial stitch removed, a larger tube passed. A larger catheter (winged) substituted.

14th.—Removed the deep sutures; wound closed except where the drain enters.

It would answer no purpose to follow out the daily notes of this case. The child had no pain nor any

trouble; her peevishness and fretfulness entirely passed away, and after the second day she became joyous and laughter loving.

26th.—Urine ceased to come by the wound, the dressing remaining dry; nevertheless I thought it wise to retain the catheter a little longer.

March 4th.—Removed drainage-tube and catheter. The child is well and has gained flesh very considerably.

The operation was performed under the carbolic spray, and the wound was dressed always with the same precautions.

Case 2.—William W—, æt. 60, came into Charing Cross Hospital under my care 20th April, 1885, with frequent and painful micturition. The man was weakly, looking older than his stated age, and said that in consequence of having to pass water frequently he had but little sleep.

I passed a sound and immediately detected a stone. The bladder felt somewhat roughened, but the prostate was very large. Although the whole manipulation was very gentle considerable hæmaturia followed and continued for sixty-three hours.

24th.—I injected the bladder and measured the stone. I succeeded in obtaining three diameters, viz. $1\frac{8}{16}$, $\frac{13}{16}$ and $\frac{7}{16}$ inch respectively. Again hæmaturia continued for some days and the man suffered a good deal after micturition. In consequence of this condition and of the large size of the prostate, I determined to perform the high operation, to prepare him for which I caused a flexible catheter to be passed daily and the bladder to be injected with a solution of carbolic acid one in sixty until a sense of distension was experienced.

30th.—When the patient was under the influence of ether the bladder was slowly filled with 16 oz. of the same solution. The catheter was plugged and a broad piece of tape tied round the penis. Notwithstanding the amount of fluid in the bladder, percussion gave clear notes down to, or very nearly down to, the pubes.

I made an incision three inches long in the middle line from above downwards and cautiously divided the linea alba and fascia transversalis. This fascia, the recti and the skin, were held apart by two broad retractors, and now placing my forefinger on the front of the bladder I pushed up the fold of the peritoneum, placed upon it a broad retractor, and confided it to Mr. Cantlie, who was assisting me. I met with no resistance whatever in thus pushing upward the peritoneum; it glided quite smoothly and easily from the anterior face of the bladder. The prevesical fat was now incised; two veins required ligature. With the edge of the knife directed downwards I made a small opening in the bladder as high as seemed desirable and placed a blunt hook in it to prevent the organ, as it emptied itself, from sinking into the pelvis; then with successive touches of the knife, the bladder was incised. The attached surface of the mucous lining was marked with large distended veins. Most of these were avoided, but two had to be tied, and three arteries in the thickness of the vesical wall were twisted. The organ was laid open to the extent of about two inches. I passed in my fore and middle fingers, and, gripping the stone between them, easily removed it. The wall of the bladder still oozed, and I was reluctantly obliged to apply a hæmostatic (one part of Liq. Ferri perchloridi to six of water). After this the bladder and other parts were sewn up and treated as in the former case.

May 1st.—The patient passed a good night, almost entirely free from pain. Temp. 99·2°. Urine came by the wound, which was perfectly healthy; it was syringed out with carbolic acid solution. A larger catheter (the one passed yesterday having become plugged) was introduced.

2nd.—Quill suture removed, wound healed except at site of drainage-tube.

4th.—Some small shreds of sloughed tissue stained with the perchloride came away. The man has had no pain nor fever.

10th.—All the wound has been healed for the last three

days save an opening that might perhaps admit an ordinary cedar pencil through which some urine flowed, though by far the largest part came by the catheter.

17th.—The opening above the pubes much smaller. He complained of some soreness in the urethra, probably produced by the catheter. This was removed on the 15th. To-day he passed urine by the urethra. The fluid, as usual in these cases, caused a good deal of scalding.

He went out quite well in the middle of July.

On 24th March, 1886, I received a note from Dr. Hughes, of Deal, who sent me this last patient, from which the following is quoted: "The old man, William W—, for whom you performed supra-pubic lithotomy, is in robust health and able to do a good day's work."

Remarks.—I would direct attention to the ease and facility with which these patients recovered, reminding the Society that this is especially the characteristic of Cheselden's, Petersen's, Günther's and other patients. My first case, the little girl, had no pain or trouble from the hour of operation. In my second case the fistula lasted some weeks. The man was somewhat troubled in mind on this account, although I was able to assure him that the opening would close within a moderate interval, which in fact it did.

The objections urged against the high operation are found, on examination, to be untenable or exaggerated. They are these:

1. The danger of wounding the peritoneum.
2. The danger of urine collecting and putrefying in the wound.
3. The danger of establishing a urinary fistula.
4. The fear that the bladder may become adherent to the abdominal wall and that thus its function may be impaired.

I propose to examine the first of these objections at the end of this communication, since it involves many points in the method of operating, in the preparation of the patient and certain matters regarding the position of the peritoneum in different states of the bladder and rectum.

These I, as well as certain other surgeons, have made the subject of experiments recorded in an appendix.

We pass on to the second objection,—the fear that urine may collect and putrefy in the wound, and with that subject we may consider the after-treatment.

Fresh urine that is not ammoniacal does no harm to a recent wound; on the contrary it is a non-irritating irrigation; and I submit that by careful use of antiseptics we can prevent it becoming ammoniacal in all parts accessible to an injection. Now, the peritoneum on the bladder leaves uncovered a triangular surface, bounded on each side by the hypogastric arteries to which it is firmly attached. This space, when the organ is distended, is (in the adult) from two to three inches long and a little more than an inch wide at its base; but when the bladder contracts it becomes very small, and as urine cannot pass beyond the lateral boundaries, all implicated parts in a properly conducted operation are perfectly within reach of an injected fluid.

But it may be questioned whether the after-treatment I adopted is the best. Such cases have been dressed in every possible manner. By T-shaped drainage-tubes and by simple meshes of hemp introduced through the wound into the unsewn bladder and with only the upper edge of the skin wound sewn; by no dressing at all save wool or tow to absorb the urine; by position, namely, on the side a little turned to the front, and many others; I do not find that the statistics of one method have any advantage over the others; yet it appears to me that by suturing the whole thickness of the abdominal wall one probably diminishes whatever tendency there may be to a subsequent hernia. Also it would seem that by stitching the bladder a more rapid healing must follow; indeed in three out of his four cases, Petersen procured primary union.

Günther says[1] that a catheter passed per urethram prolongs recovery. Other surgeons doubt this assertion.

The danger of establishing a urinary fistula need hardly

[1] 'Der hohe Steinschnitt,' p. 80, Leipzig, 1851.

detain us; such mishap has never, I believe, occurred. My case, No. 2, is an instance of the longest duration of such a fistula, viz. eleven weeks. It is doubtless a long period for recovery from lithotomy; but knowing as I do the state of this man's bladder and prostate, as also his weak and senile condition, I conceive that he recovered quite as quickly as he would have done from a lateral lithotomy, and that he ran infinitely less risk of not recovering at all.

The danger that the bladder may lose the power of emptying itself by becoming adherent to the abdominal wall was disproved by Cheselden in these words[1]: "Joseph Reynolds, who was cut May the twenty-second, 1722, and discharged cured, in the beginning of July, was about the middle of October following taken ill of a fever, with violent vomitings, of which he died in a few days, having enjoyed perfect health from the time of his cure to this illness. I opened him, and found his kidneys and bladder free from any disorder, and the wound in the bladder united with a firm, smooth cicatrix, the outside of the wound being joined to the wound made through the integuments, it was perfectly empty of water, which shows this connection of the bladder to the integuments was not inconvenient on that account; and not only this patient, but all others have been able to contain as much urine in their bladders at once, as persons that have not been cut."

The danger of wounding the peritoneum has been very much exaggerated. I am, of course, aware that this mishap has occurred to certain operators; yet my experience on the living, and my numerous experiments on the dead, subject cause me unhesitatingly to say that such misfortune can always be avoided.

The height above the upper edge of the os pubis at which the lower margin of this membrane in front of the bladder lies, varies somewhat in different individuals, even with pelvic organs equally full or empty. In chil-

[1] 'A Treatise on the High Operation for Stone,' by William Cheselden, 1723, p. 20.

dren it is practically out of the way, as in my case of Rose A— (see also Appendix).

In some adults when both bladder and rectum are empty, this fold lies a line—occasionally even two lines—below the upper margin of the pubes; but it more commonly lies above, even considerably above, that bone; but wherever it may be while the bladder is empty, it always rises when from 6 to 16 oz. of fluid are injected, and that to a height quite compatible with a safe high lithotomy.

A device for pushing up the bladder still further, namely, distension of the rectum with an india-rubber bag, was devised and practised by Dr. Petersen, of Kiel,[1] It may be granted that when the true pelvis becomes thus forcibly occupied by a foreign body, the bladder will to some extent be extruded, a change which as Dr. Garson[2] has shown can only take place by stretching—even to double its length—of the prostatic urethra; a process which can hardly be free from danger in elderly persons.

But I am able to state from a series of experiments, in twelve only of which were perfectly accurate measurements taken, (see Appendix), that distension of the rectum makes but very little difference in the position of this peritoneal fold; never more than a quarter of an inch, oftener an eighth of an inch, and sometimes its elevation was barely perceptible.

I fear I must also say that Dr. Petersen must have been misled in his experiments by a faulty method of procedure. A glance at his table will show this, since he assigns to this fold a position which is anatomically impossible. Out of ten cases there were seven in which this fold is stated to lie at two finger-breadths, and in one case at three finger-breadths below the pubes, that is to say at least one and a half and two and a quarter inches respectively.

This slight influence of rectal distension is confirmed by Dr. Garson's experiments. Table II, p. 356 is a copy

[1] 'Langenbeck's Archiv,' vol. xxv, p. 752.

[2] 'Edinburgh Medical Journal,' Oct., 1878.

of all that part of his table which refers to this fold of peritoneum. The important portions are cases 1, 2, 3, and 6, 7, 8. Nos. 1 and 6 have the same amount in the bladder; in the former the rectum is full, in the latter empty; yet the peritoneal fold lies in both at the same level. Such is also the condition in Nos. 2 and 7, and again this fold lies at precisely the same height; while in No. 8, with a like amount in the bladder, but with an empty and contracted rectum, this fold lies six tenths of an inch higher than in any case in which the rectum is full.

My own experiments, twelve in number, are added in an appendix. In summing up their result I would say that I never found distension of the rectum raise the peritoneum more than a quarter of an inch, oftener only an eighth of an inch, and sometimes its elevation was barely appreciable; the conclusion being that distension of the rectum, though it may be dangerous, is useless in high lithotomy.

These facts being fixed, I may say a few words as to the mode of operating. A catheter should be first passed into the bladder; it may be either metallic or flexible. An operator with but little experience may prefer a metal one, as in a later stage it may serve as a guide to opening the bladder. Through the catheter the bladder is to be injected with such amount of an aseptic solution as previous experience shows the patient is able to bear. If a metal catheter be used it must be plugged; if a flexible one it must be removed; in either case if the patient be a male a fillet is to be tied round the penis; if a female a finger-tip introduced a little way per vaginam may be used to compress the urethra against the pubes, thus effectually preventing micturition; or, should it seem desirable to steady the stone, the tip of the finger may do so, while the front of the first or second phalanx will prevent escape of the injected fluid.

All incisions should be made from above downwards. To cut through the linea alba without opening the peritoneum is very easy, and is constantly done by all who

practise abdominal surgery. The fascia transversalis should be incised just above the pubes, and a director, kept close to its deep surface, passed from below upwards. The triangular interval left by the two sides of the peritoneal fold now comes into view; the bladder being concealed by a layer of very soft fat. Should the interval not be large enough a mere touch of the finger will increase it; the peritoneum lies on, without being attached to, this part of the bladder. The veins in the fat are easily seen and may as a rule be avoided.

The opening of the bladder is best begun above by a little quick thrust of the bistoury, and before carrying the incision further it is well to pass in a blunt hook behind the knife, thus obviating too rapid contraction and collapse of the organ into the pelvis.

When possible the stone should be removed with the fingers.

Were not this paper already, I fear, too long several other points might be discussed.

For female children, probably for females of all ages, whenever lithotomy is necessary the high operation is preferable to all other methods; it is quite as safe and cannot lead to any form of urinary fistula.

Lateral lithotomy in boys gives excellent results when the stone is not large; nor have I seen any of the evils alleged to result from division of the vas deferens within the prostate. I cannot, however, but think that any stone, large enough to render laceration of the prostate probable during its removal by the perineum, should be taken out above the pubes. The route to the bladder is shorter, through less important and vascular tissues, and there is no danger from hæmorrhage. The results obtained by Cheselden and by other more modern operators show the remarkable ease of recovery after a *sectio alba*.

For adult males the high operation is probably to be limited to stones of a certain size and to some cases of diseased prostate and bladder. I consider, however,

that the limit of size has been placed too high, and that a stone weighing 2 oz. is, unless amenable to lithotrity, most safely removed by the high operation. Save in a few very rare cases distension of the rectum is unnecessary. Should the peritoneum, when exposed, be found to lie very unusually low, the surgeon could introduce a bag per rectum if he deemed it desirable.

For tumours of the bladder, unless situated very close to the urethral orifice, high section of the bladder is especially indicated, and if such tumour lie at the back of the organ that portion may be advantageously raised and fixed by distension of the rectum.[1]

APPENDIX.

The objects of this Appendix are—1st, to explain why the experiments herein detailed were made; 2nd, to explain the method of conducting them.

In studying the question of supra-pubic lithotomy a very important point is the position, in different states of the pelvic viscera, which may be assumed by that fold of peritoneum lying between the anterior abdominal wall and the bladder.

In 1880 Dr. Petersen, of Kiel, published an account of ten experiments made to ascertain the relative position of the anterior fold of the peritoneum and of the upper border of the os pubis. He proceeded thus. He made "an incision through the linea alba just below the umbilicus. The position of the prevesical peritoneal fold was then estimated by the introduction of a finger."[2]

It is only fair to add that Petersen acknowledges these measurements to be not quite certain (nicht ganz sicher).

[1] A plate was handed round showing a position employed by Trendelenburg (see 'Langenbeck's Archiv,' Bd. 31, p. 514), and which the author of the present paper recommends as well adapted for exploration of the fundus of the bladder.

[2] "Ueber Sectio alba," 'Langenbeck's Archiv,' vol. xxv, p. 757.

But I fear we must go further. The professor found that when both bladder and rectum were empty this fold lay in one case level with, in another one finger-breadth, in seven two, and in one three finger-breadths below the upper margin of the os pubis.[1] Now, the average of ten measurements of five finger-breadths (index and middle) is a little over an inch and a half.

The average of ten measurements of the depth of the pubes, or, in other words, of the length of the symphysis, is one and seven tenths of an inch. Therefore, according to these experiments the fold of peritoneum lay within two lines of the margin of the pubic arch.

Considering that, however contracted, the bladder must occupy more than two tenths of an inch behind the pubes, the condition of affairs thus described appeared to me impossible; more especially when Petersen gives one case in which the prevesical fold lay three finger-breadths, *i. e.* just over two and a quarter inches below the upper margin of the os pubis, I cannot but feel great doubt as to his results, more especially as Dr. Garson says that when the bladder and rectum are empty, the peritoneal fold usually lies a few millimetres *above* the margin of the pubes.

Dr. Garson[2] has also studied this question by personal experiment, and by measurements taken from engravings of frozen subjects in Pirogoff's and Braune's Atlases. In his conclusion No. 3, he says "that the raising of the peritoneum can also be produced by simple distension of the rectum." It is to be regretted that Dr. Garson gives no instance of this. His table of thirteen cases contains none in which the bladder is empty and the rectum full. Moreover with all the diffidence that I must feel in combating the conclusions of so eminent an authority, it must be stated that his results do not tally

[1] I subjoin a copy of his table, with French converted into English measures. I may add that the average distance of the internal orifice of the urethra is under two inches from the upper margin of the pubes.

[2] 'Edinburgh Medical Journal,' October, 1878.

TABLE I.—*Dr. Petersen's (Kiel) Table, the metric system being reduced to English measure.*

Bladder, contents of, in drachms	Rectum, contents of bottle in.	1	2	3	4	5	6	7	8	9	10
0	0 oz.	− 2 F.	− 0 F.	− 2 F.	− 2 F.	− 1 F.	− 2 F.	− 2 F.	− 2 F.	− 2 F.	− 3 F.
	7 „	„	„	− 1½ F.	− 1½ F.	− ¾ F.	− 1½ F.	− 1½ F.	− 1 F.	− 1 F.	− 2½ F.
	14 „	„	+0·15 in.	− 1 F.	− 1 F.	− ½ F.	− 1 F.	− 1½ F.	0	− ½ F.	− 2 F.
	21 „	− 1 F.	+0·27 in.	− ½ F.	− ½ F.	− ¼ F.	0	− 1 F.	+0·23 in.	0	− 1½ F.
7 oz.	0 oz.	− 1 F.	+0·33 in.	− 1½ F.	− 1 F.	− ½ F.	− 1½ F.	− 1¾ F.	0	− 1¼ F.	− 1½ F.
	7 „	„	+0·43 in.	„	− ½ F.	0	− 1 F.	− 1½ F.	+0·11 in.	− ¾ F.	− 1 F.
	14 „	„	+1·28 in.	− 1 F.	0	+0·29 in.	0	− ½ F.	+0·29 in.	0	0
	21 „	0	+1·7 in.	0 almost	+0·039 in.	+0·54 in.	+0·03937 in.	0	+0·59 in.	+0·15 in.	+0·25 in.
14 oz.	0 oz.	0	+0·87 in.	− ½ F.	− ½ F.	+0·17 in.	− 1 F.	− F.	+0·23 in.	− ¾ F.	+0·1 in.
	7 „	0	+1·18 in.	„	0	+0·54 in.	0	− ½ F.	+0·54 in.	0	+0·3 in.
	14 „	+0·59 in.	+1·85 in.	0	+0·039 in.	+0·88 in.	+0·3937 in.	0	+0·7 in.	+0·15 in.	+0·5 in.
	21 „	+1 in.	+2·27 in.	+0·23 in.	+0·23 in.	+1 in.	+0·59 in.	+0·29 in.	+1 in.	+0·24 in.	+0·8 in.
21 oz.	0 oz.	+0·33 in.	+1·49 in.	0	0	+0·669 in.	+0·29 in.	− 1 F.	+0·39 in.	0	+0·6 in.
	7 „	+0·59 in.	+1·8 in.	+0·12 in.	+0·039 in.	+0·88 in.	+0·3937 in.	0	+0·7 in.	+0·12 in.	+0·8 in.
	14 „	+1 in.	+2·4 in.	+0·39 in.	+0·54 in.	+1·18 in.	+0·7 in.	+0·29 in.	+0·8 in.	+0·24 in.	+1·2 in.
	21 „	+1·33 in.	+2·5 in.	+0·87 in.	+1 in.	+1·35 in.	+1 in.	+0·87 in.	+1·3 in.	+0·59 in.	+1·3 in.

with his deduction. Cases 1 and 2 have the bladder distended and have also the rectum "much distended." Now, in them the distance of this fold above the pubis is given as forty and fifty-five millimetres; but Case 3 with an equally full bladder, but with a rectum only "moderately distended," the distance is fifty millimetres *i. e.* two tenths of an inch less than Case 2, and four tenths more than Case 1. Evidently in these three cases rectal distension had no effect.

TABLE II.—*Garson's Table, the metric system being reduced to English measure.*

No. of case.	Condition of bladder.	Condition of rectum.	Distance of peritoneum above pubes.
1	Much distended[1]	Much distended[2]	1·57 inch.
2	Fully distended	Fully distended	2·16 „
3	Much distended	Moderately distended	1·96 „
	Cases where Bladder and Rectum were empty.		
4	Almost empty	Absolutely empty and contracted	0·19 inch.
5	Absolutely empty	Almost empty	0·. „
	Cases with empty Rectum and distended Bladder.		
6	Much distended	Absolutely empty and contracted	1·57 inch.
7	Distended	Empty and contracted	2·16 „
8	„	„ „	2·75 „
9	Half filled	Empty	0·079 „
	Cases with moderately distended Bladder and Rectum.		
10	Moderately full	Moderately full	0·3 inch.
11	Half full	Half full	0·79 „
12	Moderately full	Moderately full	0·62 „

[1] Bladder filled with 8 oz. 3 drs. (240 grammes of fluid.)

[2] Rectum distended by bag containing 10½ oz. (300 grammes) of fluid; its circumference being 9·84 inches (25 cm.).

Again, when we compare Cases 1, 2, and 3 with Cases 6, 7, and 8,[1] we find the following. The former series are, as stated, cases with distended bladder and rectum; the peritoneal fold lies forty, fifty-five, and fifty millimetres respectively above the pubes. Cases 6, 7, 8 have the bladder equally distended, the rectum "empty and contracted." The fold lies forty, fifty-five, and seventy millimetres above the pubes, that is on an average seven millimetres higher when the rectum is empty than when it is full. In the table subjoined I have reduced these measures to inches—seventy millimetres is two and three quarter inches, and this was obtained when the bladder was distended (not "much distended") and the rectum empty and contracted.

Surely these cases, 6, 7, 8, show, when compared with Cases 1, 2, and 3, that distension of the rectum has no effect on the anterior fold of the peritoneum.

Wishing to gain a definite insight into the true state of the case with regard to this fold I instituted a series of experiments the result of which is subjoined. They were conducted in the following manner:

The bladder was emptied by the catheter and the rectum by washing it out with water. An incision was then made through the linea alba and fascia transversalis. The position of the lower border of the prevesical fold was measured.

In Series I the bladder only was filled with varying amounts of water.

In Series II the bladder was filled first, then the rectum.[2]

In Series III the rectum was filled first and then the bladder; subsequently, while the bladder was still full, the rectum was emptied.

In each one of these different conditions the position of the fold was carefully noted.

[1] In Garson's table, Case 7, the particular point in question is not marked. I have therefore omitted it and changed (after 7) the numbering.

[2] By means of a distensible india-rubber bag and a Higginson's syringe.

TABLE III.—*Experiments (Barwell).*

Cases in which Bladder only was filled.

No.	Subject.	Age.	Bladder.	Rectum.	Relation of fold to pubes.
1	Child	2½	Empty	Empty	⅛ inch above
			2 oz.	,,	1½ ,, ,,
2	Child	4	Empty	Empty	⅛ inch above
			4 oz.	,,	1⅜ ,, ,,
3	F.	62	Empty	Empty	⅛ inch below
			8 oz.	,,	¼ ,, above
			16 oz.	,,	2 ,, ,,
4	M.	39	Empty	Empty	¼ inch above
			10 oz.	,,	1½ ,, ,,
			16 oz.	,,	2½ ,, ,,
5	M.	32	Empty	Empty	Level
			10 oz.	,,	1 inch above
			14 oz.	,,	1½ ,, ,,

In which Bladder was filled first, then Rectum.

6	M.	30	Empty	Empty	⅛ inch above
			6 oz.	,,	⅜ ,, ,,
			10 oz.	,,	1⅛ ,, ,,
			15 oz.	,,	1 1/16 ,, ,,
			Empty	12 oz.	⅛ inch above
			6 oz.	,,	1 ,, ,,
			10 oz.	,,	1⅜ ,, ,,
			15 oz.	,,	2⅛ ,, ,,
7	M.	34	Empty	Empty	⅛ inch above
			10 oz.	,,	⅜ ,, ,,
			10 oz.	10 oz.	1 3/16 ,, ,,
8	M.	35	Empty	Empty	⅛ inch above
			10 oz.	,,	1⅜ ,, ,,
			16 oz.	,,	2⅜ ,, ,,
			16 oz.	10 oz.	2⅛ ,, ,,
9	M.	32	Empty	Empty	Level
			12 oz.	,,	1 5/16 inch above
			12 oz.	12 oz.	1 5/16 ,, ,,

In which Rectum was filled first, then Bladder.

No.	Subject.	Age.	Bladder.	Rectum.	Relation of fold to pubes.
10	M.	72	Empty	Empty	$\frac{1}{4}$ inch above
			,,	8 oz.	$\frac{1}{4}$,, ,,
			8 oz.	8 oz.	1 ,, ,,
			16 oz.	8 oz.	$2\frac{1}{4}$,, ,,
			16 oz.	Empty	2 ,, ,,
11	Boy, immature and small	14	Empty	Empty	$\frac{1}{8}$ inch above
			,,	6 oz.	$\frac{1}{8}$,, ,,
			2 oz.	6 oz.	$1\frac{1}{8}$,, ,,
			4 oz.	6 oz.	$2\frac{1}{16}$,, ,,
			4 oz.	3 oz.	$2\frac{1}{16}$,, ,,
			4 oz.	Empty	$1\frac{1}{8}$,, ,,
12	—	—	Empty	Empty	$\frac{1}{16}$ inch below
			,,	10 oz.	Level
			10 oz.	10 oz.	$\frac{1}{8}$ inch above
			13 oz.	10 oz.	$1\frac{1}{8}$,, ,,
			16 oz.	10 oz.	$1\frac{1}{4}$,, ,,
			16 oz.	Empty	$1\frac{1}{8}$,, ,,

The conclusion seems inevitable that distension of the rectum produces no such elevation of the peritoneum as would be of any value to the operator, nor does it appear that there is any object to be gained in lithotomy by pressing forward the back of the bladder, as a stone, unless very small, is quite within reach. If cystotomy be performed for the removal of a growth the rectum should certainly be distended.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' New Series, vol. ii, p. 94.)

A CASE

OF

ENCYSTED VESICAL CALCULUS OF UNUSUALLY LARGE SIZE

REMOVED BY SUPRA-PUBIC CYSTOTOMY.

BY

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Thomas K—, æt. 61, soldier, was admitted on the 13th January, 1885, into the London Hospital, suffering ostensibly from stricture and cystitis. While in the army, from which he had retired with a pension, he had served in various parts of the world, including the Crimea. He had been treated for stricture in Ceylon. He had not worked for two years. For sixteen years he had suffered from occasional stoppage of the water, combined with considerable pain in the loins and at the end of the penis. For six years there had been slight hæmorrhages at times. Latterly he had failed in health and lost flesh, and the urinary complaint had become more troublesome. On admission he complained of not being able to hold or pass

his water properly. The bladder was very irritable. Signs of cystitis were present, the urine being ammoniacal and containing pus. There was not more albumen than the pus would account for.

The bladder was washed out, at first with a weak solution of carbolic acid (1 in 400), and afterwards with iodoform in mucilage, and he was ordered some infusion of buchu and tincture of hyoscyamus three times a day, as well as two drachms of confection of senna to be taken every morning. Under this treatment, combined with rest, he improved. The pain diminished in severity, the bowels acted better, and he was able at times to pass his urine more naturally.

On examination per rectum a large round smooth swelling, very firm and hard, was felt anteriorly in the situation of the prostate gland, and suggested either an unusually enlarged prostate or the presence of a prostatic calculus. Nothing could be detected, either in the prostatic urethra or in the bladder, by means of the sound. The patient was asked to make water into a porringer, and the stream was found to drop from the end of the penis, as it does in cases of enlarged prostate. It was decided to advise an examination under an anæsthetic, and a median urethrotomy for the purpose of exploration and subsequent drainage of the bladder, any further procedure being dependent on the result of the examination. The patient gave his consent to any procedure that might be considered desirable.

On the 24th of February he was taken to the operating theatre and anæsthetised. Nothing could be detected with the sound. A grooved staff was then passed into the bladder, and, the patient having been placed in the ordinary lithotomy position, an incision about an inch long was made in the middle of the perineum, and the membranous urethra was opened in front of the prostate. Exploration with the finger failed to detect anything abnormal in the prostate, but it was ascertained that the hard, rounded mass was not connected with the

prostate, and that it was covered by the left wall of the bladder, which was pushed towards and even beyond the median line. It was also found that the mass overlapped the prostate and that the finger placed in the rectum could be pushed between it and the prostate gland, which was not at all enlarged. By supra-pubic examination it was evident that the mass was of considerable size, and not very moveable, and it became a question whether it was an encysted calculus or a growth from the pelvic walls. By further examination with the sound pushed in up to the hilt, a stone was struck far back in the bladder, and with a pair of lithotomy forceps I succeeded in grasping the end of the stone without being able to shift its position. It now seemed evident that I had to deal with a calculus or calculi either in a pouch or in a separate division of the bladder, and I determined to open the bladder above the pubes.

Having released the patient from the lithotomy position I passed a well-curved staff into the bladder, and made an incision in the middle line above the pubes about three inches in length, and carefully divided the structures close to the pubes until I could feel the point of the staff through the bladder wall. My colleagues, Mr. Reeves and Mr. E. H. Fenwick, assisted me. The bladder was reached and opened above the pubes, and the opening cautiously enlarged, chiefly downwards. A vein in front of the bladder, which has been named by Mr. Fenwick the inverted Y vein, was divided and tied. A loop of silk was passed through the bladder wall on each side, to enable my dressers to hold aside the edges of the vesical wound and to steady the bladder. The end of a stone could now be felt and seen to pass out of an aperture towards the back of the bladder. It was grasped with forceps, but very little impression was made on its position, even after passing a lithotomy scoop between the calculus and the wall of the pouch in which it lay. Lithotrites were useless. Under these circumstances there were two alternatives, viz. either to abandon the operation

or to break up the calculus. It occurred to me that division of the calculus might be effected with a chisel and mallet, and I decided to make the attempt. As the calculus below was perfectly smooth and fitted well into the pelvis, I did not think that any injurious bruising of the base of the bladder would result from the concussion of the stone, and I guarded against this by introducing a lithotomy scoop between the calculus and the wall of the pouch, and supporting the calculus during the taps of the mallet by resting the handle of the scoop against the wall of the abdomen and using it as a lever of the first kind. The chisel cut the stone readily enough, and severed it into several large fragments, more or less wedge shaped, which were extracted piecemeal.

There was one circumstance which I had not anticipated, viz. free oozing of blood from the congested mucous membrane of the bladder and its pouch during the manipulations for breaking up, and removing the segments of the calculus. Another event was the escape from the pouch, as soon as the stone had been shifted, of a quantity of most fetid urine. After the removal of the last portion of the calculus the bladder and its pouch were carefully washed out with an antiseptic solution, and all ascertainable fragments were removed. A few chips, however, escaped detection, doubtless having been enveloped in blood-clot. At the suggestion of Mr. Fenwick I sewed up the wound in the bladder, using fine silk introduced with the glover's suture, and a second suture was introduced at the lower angle of the vesical wound. The recti muscles were united with interrupted sutures, and lastly the skin and fascia. In order to guard against urinary infiltration, a drainage-tube was inserted between the lips of the superficial wound, reaching down to the anterior surface of the bladder. A silver tube without a sponge was inserted into the bladder through the perineal wound, the supra-pubic wound was dressed with cotton wool and gauze, and the patient was sent to bed. The operation had lasted an hour and a half. The patient was

not so much exhausted by the operation as was expected, nor did his temperature show any marked rise during the first twenty-four hours. He complained of wind and some pain. A hypodermic injection of morphia was given. The urine passed freely through the tube. He was not sick, and was able to take milk and brandy mixture.

26th.—Patient passed a fairly good night, sleeping for some hours. Very little pain; sensation of fulness in the bladder; forty-five ounces of urine were collected. Pulse 100, temp. 99°. Bladder washed out with solution of thymol.

On February 28th I found an extending red blush at the edges of the wound, and the drainage-tube displaced. Believing that this must be caused by some pent-up discharge, probably mixed with urine, I opened up the wound, and having mopped out some urinous fluid mixed with pus, powdered the surface of the cavity with iodoform, and covered it with cotton wool. There had been a free discharge of urine by the perineal wound through the drainage-tube amounting to forty-two ounces of collected urine during the twenty-four hours. The temperature was 99°, and pulse 96. Milk, beef-tea, and brandy and egg mixture were taken freely. The surface of the wound cavity above the pubes was sloughy, and underwent a gradual process of removal of slough and granulation. It was cleansed daily, irrigated, and powdered with iodoform.

The notes on the 3rd of March, state: "Very restless night, acute pain at times, smell of upper wound very fetid. Thick grey slough on surface, and some surrounding inflammation. Patient wanders slightly and picks at the bedclothes. No vomiting and no sign of peritonitis." During the next few days he improved materially, and the wound began to granulate healthily after the separation of the slough.

On the 9th the silver lithotomy tube was removed from the perineal wound, and a large india-rubber tube was

substituted. Most of the urine came away below, but occasionally some would well up behind the pubes. Patient was no longer delirious. His temperature was normal and his pulse 80. One of the ligatures came away in the silver tube with some thick matter and slough.

16th.—Patient slept seven and a half hours last night; thirty ounces of urine collected in the night, sixty ounces altogether in the twenty-four hours. A long slough in the tube.

19th.—Very restless. Has had very little sleep. Tube got blocked up with slough or membrane, and the urine ran over the pubes. The tube was taken out and cleansed. A soft flexible catheter was introduced above the pubes and withdrew a large quantity of foul urine. Great pain in right lumbar region. Temperature 101·5°. I had to make an opening in the scrotum for drainage as a pouch had formed there containing urine.

20th.—Much better. Temperature normal. Pain abated. Tongue clean. Pulse 80. Being very anxious to be allowed to be out of bed, and confident that he would benefit by the change, he was placed in a chair and wheeled about for half an hour or an hour.

April 1st.—Since the last note he had been going on well, passing a fair amount of water by the tube. The anterior wound was gradually closing, and was syringed out daily with thymol solution. The bladder was also washed out, the solution running freely through the perineal tube. He slept fairly well. His appetite had improved and he took meat and potatoes.

On the 17th the house surgeon, who with Mr. Haynes, the dresser, had been very attentive to the patient, finding that the abdominal wound had closed over the aperture leading to the bladder, withdrew the perineal tube. I had intended retaining the tube till the wound had soundly healed, but when I saw the patient in the afternoon the perineal opening had contracted so much that I could not have reintroduced the tube without placing the patient under an anæsthetic, and, as I thought

that this might possibly do him more harm than the tube would do good, I reluctantly abandoned the tube altogether. The patient was now in very fair condition, able to walk and pass his water with a considerable jet, and he was extremely proud of his capabilities in this matter. Unfortunately a little grit, part of the remaining *débris* of the calculus became impacted in the urethra, and the obstruction caused the passage from the bladder to the wound above the pubes to reopen so as to again admit a small catheter. If the tube had been retained, according to my instructions, this would not have occurred, and the opening would have soundly closed. At this time the patient had practically recovered from the operation. He sat up daily, took his food well, his urine was clear, and on warm days he went into the garden in a chair.

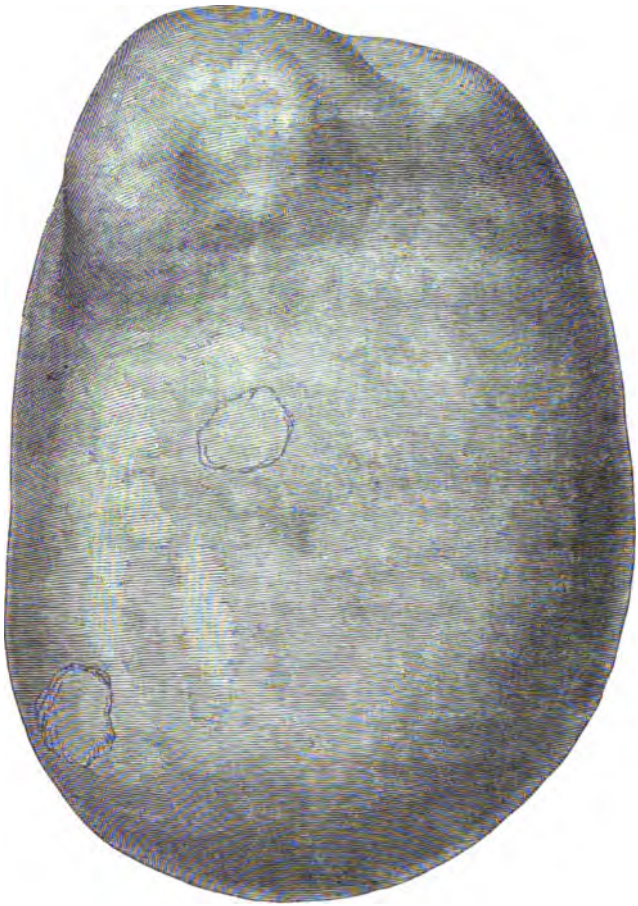
Early in May the supra-pubic wound had nearly healed, leaving only a small fistula. The patient was kept in the hospital because I was anxious to close the opening, and for this purpose his water was drawn off with a flexible catheter two or three times a day.

About the middle of May he fell down in the ward, and, as he felt fatigued with being up so long, and was not gaining strength, I advised him to remain in bed during the greater part of the day, draw off his water, and see if the fistula would close.

At the end of May a fresh attack of cystitis developed. His urine became strongly alkaline, turbid, and ammoniacal, and contained pus. There was a considerable discharge of pus from the opening above the pubes, and an abscess formed and opened over the tendon of the adductor longus in the right thigh. His appetite failed. Diarrhœa set in. Exploration of the region of the wound disclosed some bare bone near the symphysis. He became comatose, and died on June 4th, more than three months from the date of operation. With considerable difficulty I obtained permission to inspect the abdomen only, and this limited post-mortem was performed on the 5th of June.

Post-mortem.—The bladder was fairly capacious, and its walls were thickened from muscular hypertrophy. Coming off from it behind and above the trigone by a rounded opening was the large pouch in which the stone had been contained. This ran first outwards and then forwards, and when distended reached beyond the margin of the prostate gland. Its walls were thick and comprised the mucous, muscular, and fibrous coats of the bladder. The left ureter was closely connected with the pouch, winding round it and externally appearing to terminate in it; but a bent probe passed from above downwards through the left ureter, was seen to emerge by the side of the trigone of the bladder proper. From the lateral position of the pouch parallel to the bladder, from the left wall of the bladder running directly backwards from the middle of the prostate, from the collection of ammoniacal urine in the pouch found at the operation, and from an evident filling of the pouch afterwards, I had thought it not improbable that the pouch was an integral portion of the bladder. The mucous membrane of the bladder and pouch was inflamed, and the ridges were coated with muco-pus mixed with phosphates. The edges of the wound in the bladder were puckered, coated with phosphatic muco-pus, and firmly adherent to the posterior surface of the pubes. An opening which had enlarged slightly by ulceration during the last few days of life led to the surface, and also by means of a branching canal to the perineum and to the opening in the right thigh. The left pubic bone was bare of periosteum and superficially necrosed. There was an abscess deep in the perineum on the right side. Most of these changes occurred at the latter end of May and the beginning of June. The kidneys were of unequal size. The right kidney was larger than the left and larger than a normal kidney. It appeared healthy, but had some cysts on its surface. On cutting into the left kidney some thin purulent matter escaped from a small cavity in the cortex, and there was evidence of interstitial nephritis running on to suppurative nephritis. The cap-

sule did not strip off readily, and the organ was puckered. The pelvis of the left kidney was slightly enlarged as well as the upper part of the left ureter. The right ureter



Calculus extracted; natural size.

was normal. The calculus when removed from the bladder was weighed by Mr. Fenwick. Excluding a considerable quantity of lost *débris* its exact weight in the moist state was 23 oz. 2 drachms and 17½ grains avoirdupois. The

nucleus weighed 65 grains. The fragments being stained of a dark colour the stone appeared to be composed of lithic acid and lithates, but in reality it is composed of fusible phosphates. After the operation the large segments were most skilfully put together by Mr. Taylor, the museum assistant at the Medical College. The stone now weighs, without nucleus and lost *débris*, 22½ oz. avoirdupois. A section has been made and shows a large cavity in the centre of the calculus due to the lost *débris*. The correct weight of the calculus must therefore be regarded as exceeding 23 oz., or 1 lb. 7 oz. avoirdupois. The dimensions are as nearly as possible 4¾ inches long, 3½ wide, and 3 inches in thickness; its larger circumference 13 inches and its lesser 10 inches. The size of the pouch may be inferred from the size of the stone, which exactly filled it, and the size of the orifice of the pouch from the size of the base of the projection from the stone. The orifice through which the stone had to be extracted was about the size of half a crown.

Remarks.—With regard to the size of the calculus there are a few instances of larger vesical calculi on record, some removed from the bladder after death and some during life. To the post-mortem category belong:

1. The calculus seen by Morand weighing 6 lbs.
2. The calculus seen by Deschamps weighing 51 oz.
3. The well-known phosphatic calculus 44 oz. in weight, and measuring in circumference 16 inches by 14, which Cline attempted to remove from Sir Walter Ogilvie, who died on the tenth day.[1]
4. The lithic acid calculus, now in the pathological museum of the University of Cambridge, measuring 15 by 13½ inches in circumference, and weighing 32 oz. 7 drachms, originally 33 oz. 3 drachms and 36 grains troy. The stone was taken from the wife of Thomas R—, a locksmith in Bury, after her death, by Mr. Gutteridge, a

[1] 'Catalogue of Calculi (Part I, H$_2$, p. 116) of Museum of Royal College of Surgeons of England.'

surgeon of Norwich, and was presented to Trinity College, Cambridge, by Mr. Samuel Battley, who was M.P. for Bury and had possession of the stone after the woman's death.[1]

5. The uric acid calculus, weighing 25 oz., and measuring 4½ inches in its long axis by 3½ in its short, and in circumference 12½ by 10½ inches, taken from the body of Sir Thomas Adams, who died on February 24th, 1667, at the age of eighty-one. The stone remained in possession of the family for years and was ultimately presented to the museum of St. Thomas's Hospital.[2]

6. A case has been recorded by Mr. Paget, of Leicester, in which a stone weighing 27 oz. was removed after death from the bladder of a woman forty-seven years of age. It was accompanied by innumerable small calculi some as large as peas and others smaller. The large stone was of a light ash colour, rough on its surface, and of a flattened oval shape. It had occasioned prolapse of the bladder, the viscus covered by the vaginal mucous membrane protruding between the labia. The external surface of the calculus was marked by a sulcus occasioned by the pressure of the distended labia pudendi.[3]

To the category of large stones removed during life belong:

7. Uytterhoeven's calculus, the cast of which measures 16½ by 12½ inches in circumference. The patient lived eight days.[4]

8. A calculus reported on the authority of Dr. W. B. Hunter, of Londonderry, as having been removed by Surgeon Joseph Hunter, I.M.S. The patient was a native in the Madras Presidency; the supra-pubic operation was performed, the stone weighed 25 oz., and the patient lived three days.[5]

[1] Dr. G. M. Humphry, 'Lancet,' July 25, 1885.

[2] Pathological Society's 'Transactions,' vol. xxi, p. 267. A woodcut showing the size of the stone is given.

[3] 'Lond. Med. and Phys. Journ.,' vi, p. 391.

[4] Erichsen, 'Surgery,' vol. ii, p. 986.

[5] 'Lancet,' Jan. 16, 1886.

A calculus has lately been reported to the Northumberland and Durham Medical Society as having been removed by Dr. Morrison from a sailor, æt. 52, and weighing 1lb. 6¾ oz. (whether troy or avoirdupois is not stated). In the report this is euphemistically styled "the largest stone ever removed during life." It is, however, not quite equal in weight to my own. The composition and dimensions of the calculus are not given in the account which I have seen. The patient lived twelve days and then died suddenly. No post-mortem examination was permitted.[1]

Among calculi of smaller size the most noteworthy was one which Sir H. Thompson removed by supra-pubic cystotomy on the 29th April, 1885, from a man æt. 62. It was a uric acid calculus weighing 14 oz. avoirdupois, measuring 4⅓ inches in length by 3 inches in breath and circumferentially almost 12 inches by 8 inches. The patient made an excellent recovery.

1. It will be observed that the case stands by itself in this particular that the calculus was contained in a pouch from which only a small projecting process protruded. This rendered the operation far more tedious and difficult than any of the other recorded operations for large calculi, as the calculus had to be broken up through a comparatively small aperture and removed piecemeal. Great care had to be exercised not to damage the bladder by contusion or perforation, and there was free oozing of blood from the congested mucous membrane whenever the calculus was disturbed. Extraction of the segments was also not a very easy matter.

2. It may fairly be asked would it have been better to leave the calculus alone when its exact position was made out, or was it better to attempt extraction and carry it through? Against leaving it the following considerations

[1] Since this paper was read Mr. Thomas Smith has presented to the museum of the Royal College of Surgeons a cast of a calculus, weighing 24½ oz., which he successfully removed by the supra-pubic operation from a male patient.

may be adduced. The patient's health was failing from the presence of the calculus and its projection into the bladder proper. He was suffering pain from the calculus whenever he took exercise. He had chronic cystitis with occasional hæmorrhages. The urine had become decomposed and ammoniacal, and ammoniacal urine pent up in the pouch was a constant source of contamination to the freshly secreted urine. He could not pass his water properly, and the left kidney was being damaged by interstitial nephritis. The disadvantages of removing it were that it subjected the patient to a long and difficult operation not free from danger. The difficulties were surmounted satisfactorily, but the main disadvantage of removing the calculus consisted in the fact that the pouch in which the stone was lodged had to be left behind, and would necessarily form a receptacle for urine, and would never, perhaps, be properly emptied. At the time, however, it was not clear whether the compartment containing the stone was a hernial pouch, or whether it was an integral part of the bladder and received the left ureter. Undoubtedly if a patient enjoying good health was known to have a large encysted calculus which gave rise to comparatively little inconvenience or urinary disturbance I should not be inclined to advise interference, but when it has begun to emerge from the pouch and has become the occasion of constant pain, cystitis, and decomposition of urine the question of interference may fairly be entertained. If the pouch could be removed a great advantage would be gained. It did not occur to me to ascertain if this would have been feasible in my own case. If attempted it would, I think, have to be done from inside the bladder by inversion of the pouch and either ligature or excision and suture of the wound.

3. With regard to the details of the operation a few remarks are necessary.

(*a*) The valuable addition to the supra-pubic operation, for which surgeons are indebted to Garson and Petersen, could scarcely have been applied in the present case, owing to the perineal opening and the size and situation of

the stone. By keeping close to the pubes I avoided the risk of wounding the peritoneum.

(*b*) Seeing how readily a calculus may be broken up by means of a chisel and mallet, I think that the same method might be adopted wherever a calculus has attained so large a size that it cannot be extracted entire without risk of tearing the peritoneum, or unduly bruising or lacerating the bladder and enlarging the vesical wound. A very large calculus would almost certainly prove to be phosphatic. It is not difficult to guard against injuring the bladder walls in the process, and the chief objection lies in the risk of leaving some small chips behind to cause irritation or act as the nuclei of future stones. This risk is greater where there is a pouch than where the calculus is free in the bladder itself.

(*c*) Sewing up the bladder wound was done rather tentatively than from absolute conviction of its certain utility. To guard against danger from escape of urine, if the sutures should prove inefficient, a drainage-tube was placed in contact with the sutured opening. Doubtless the necessary contusion of the edges of the wound during the long operation prevented immediate union of any considerable part of the wound. Whether any part of the wound united in consequence of the sutures I cannot say. The sutures themselves separated and were discharged, one through the silver tube, and the other through the external wound, after being for some time adherent. I am inclined to think that the stitches did no good, but rather the reverse, as their retraction determined more sloughing of the edges of the vesical wound, and in another case I should not suture the vesical wound unless I had a clean cut to deal with which had not been subjected to any bruising. I think also that the stitches determined the adhesion of the opening to the posterior surface of the pubes.

There is another method of dealing with the wound in the bladder which might in some cases be advisable, and that is stitching its edges to the edges of the superficial

wound. I am not sure that this might not have been preferable to the course which I actually adopted. It would effectually guard against extravasation of urine and would permit the bladder to be thoroughly washed out.

(*d*) I am convinced that the perineal tube was of primary importance to the patient in this case, and I regret that I did not reinsert it after it had been removed prematurely by my house surgeon. It gave exit to the thick pus and a few pieces of slough which came away from the bladder after the operation. It drew off the major portion of the daily urine, only a little occasionally running off by the upper wound. It allowed the bladder to be washed out, and it prevented accumulation of the urine in the pouch. Hitherto surgeons have regarded infiltration of urine as one of the two chief risks of the supra-pubic operation, and deaths have not unfrequently resulted from this cause. Sir Henry Thompson, who has had marked success with this operation, thinks that there is very little risk of infiltration in ordinary cases, unless there be interference with the cellular connections low down between the anterior surface of the bladder and the pubic arch. In such cases, and in exceptional cases like my own, I believe that the insertion of a large soft tube in the bladder through a median perineal opening will prove more efficient than keeping a catheter in the bladder or inserting a drainage-tube above the pubes, and not only add nothing to the risk of the operation but will contribute materially to ensure the safety of the patient.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' New Series, vol. ii, p. 94.)

A CASE

OF

SUPRA-PUBIC LITHOTOMY,

WITH

REMARKS ON THE OPERATION.

BY

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Received March 23rd—Read March 30th, 1886.

A. F—, æt. 19, an Essex labourer, was sent to the writer at Guy's by Dr. Day, of Harlow, January 21st, 1886, for stone in the bladder. Irritability of the bladder, day and night alike, had been present all his life; symptoms of stone had been well marked for over five years, and for the last twelve months cystitis had been present. A stone was readily felt at the neck of the bladder; so closely did it fit here, and so readily did it return after being pushed away, that considerable elevation of the pelvis was required before a lithotrite could be made use of. Both this and the sound gave evidence of more than one stone.

It was decided to perform lithotomy rather than lithotrity on account of the multiple calculi, the hardness of

one calculus, and its constant position at the neck of the bladder. As to the size of the stone, this, as appeared later, had been twice correctly gauged as a very moderate one, one and a quarter inches in its long diameter. With regard to this the writer was inclined to think that this was the short diameter owing to the rectal examination giving the impression of a larger stone. Both in this case and in one in which Prof. Humphry performed supra-pubic lithotomy for a stone which actually weighed about 1½ oz., the coats of the bladder, no doubt thickened, somewhat closely embraced the stone, and thus gave an impression that the latter was larger than it really was.

Lithotomy being decided on, the supra-pubic operation was preferred on account of the age of the patient, the fact that the symptoms of calculus had certainly lasted over five years, and perhaps throughout life, that thus it was not improbable that the structure of the kidneys was impaired, and if so, it seemed reasonable to think that an incision made into the anterior surface of a bladder distended with antiseptic fluid and brought safely into reach would be more successful, in the long run, than one into the neck of the bladder, with its complicated surroundings and far more abundant vascular and nervous supply.

January 30th.—The operation was performed on the lines laid down by Sir H. Thompson in his recently published book. The patient being under ether, one of Sir H. Thompson's bags was introduced well coated with eucalyptus and vaseline, pushed quite above the sphincters and then distended with 10 oz. of warm water; 8 oz. of warm carbolic acid, 1 in 100, were then introduced into the bladder, the catheter withdrawn, and a Jaques' india-rubber catheter tied round the penis. The bladder could now be both seen and felt distended for two thirds of the distance between the umbilicus and pubes. An incision, three inches long, was then made in the middle line down to the symphysis. After division of the linea alba and fascia transversalis, an abundant layer of fat with veins bulged up into the lower angle of the

wound; this being carefully torn through with a director, the anterior surface of the bladder, pink-red and showing clearly detrusor fibres, came into view. The peritoneum was never seen, and could only be very indistinctly felt. A tenaculum being passed into the bladder, and a scalpel introduced at this spot, the left index was inserted and at once felt a stone; the opening being dilated with the other index finger the stone was removed between them. In this dilatation the bladder was felt to tear readily, but without hæmorrhage. Careful and repeated search, including the parts of the bladder behind the pubes and behind the prostate, failed to detect the other calculi whose existence was suspected. While it appeared at the time that the bladder cavity was immediately under reach, and that every part had been explored, the writer thinks that his failure to detect the two other calculi, which were, after all, present, arose from the bladder being full of the antiseptic fluid. The writer being desirous that, as the bladder emptied itself over the recently cut tissues, the first flow should be of antiseptic fluid, he allowed too little fluid to escape during the exploration. The weight of the two smaller calculi, when in fluid, must have been very slight, and stones so small and so light may have been easily displaced in currents set up in so much fluid, and thus rendered very difficult to find and seize. The only other explanation which occurs to the writer is that 10 oz. of fluid in the rectal bag may not have been sufficient to raise the bladder evenly and completely, and thus some depression or pouch may have been temporarily formed, and not reached by the finger.

No attempt was made to unite the wound in the bladder owing to the previous cystitis and the somewhat prolonged examination; two deep stitches (carbolised silk) were placed in the linea alba and two more superficially.

There was never the slightest sign of extravasation or cellulitis, but the healing was retarded by an attack of pneumonia following the operation, and due, in part, to

the ether, and, in part, to the bitter weather of this winter.

On the fourth day the wound and urine were ammoniacal, and this lasted for thirty-six hours, but yielded at once to washing out the bladder with Thompson's fluid.

Two weeks after the operation and when the wound was rapidly granulating up the patient felt as if he was passing water per urethram. It was then found that considerable hæmorrhage had taken place both from urethra and wound. It was venous in character and was readily stopped by the introduction of a small bit of sponge, well powdered with iodoform and steel sulphate, pushed firmly down into the wound. A few hours later, on the removal of the sponge, a small smooth calculus was found in the lower angle of the wound.

Two days later a second but much smaller hæmorrhage took place—yielding at once to ice—and a second small calculus came away.

Three weeks after the operation 5 or 6 oz. of urine were passed naturally, this quantity gradually increasing till the fifth week, when all the urine was passed the right way.

Remarks.—While the above case cannot be considered such a good test of the value of the operation as one in which a larger stone and an older patient are dealt with, it yet presents some features of interest. The immunity from any symptom of cellulitis or extravasation from first to last was absolute; in fact, local inflammatory symptoms were never present; there was a little tenderness the first night around the wound, but this was all.

The ammoniacal condition of the wound on the fourth day was due, in part, to the previous cystitis, and, in part, to the fact that just at this time the patient was suffering from pneumonia; he was dull and apathetic, and when turned on to his side sank as far as possible on to his back again. The way in which this ammoniacal condition yielded at once as soon as the fluid which bears

Sir H. Thompson's name was used, saturated boracic acid solution having been used for thirty-six hours without good result, was very noteworthy.

The hæmorrhage which occurred can in no way be put down to the operation. It was due entirely to the writer having failed to find the two smaller calculi. As these made their way out through tender granulations, still at that time bathed in urine, they easily caused considerable bleeding.

A few of the most important points connected with the operation will now be considered.

The distension of the rectum.—Care should be taken that the bag used for this purpose be of sufficient strength. M. Guyon[1] mentions one case in which the bag being of thin india rubber did not support the bladder sufficiently firmly, and in which the organ, yielding on this account to the pressure of the fingers, was difficult to open. In other words a thin india-rubber bag will raise the bladder but not support it steadily when it is cut down upon.

The bag, well coated with eucalyptus and vaseline, and introduced in a folded state above the sphincters, is slowly distended by means of its tube and a syringe with about 12 oz. of tepid water. Sir H. Thompson gives the amount as "12 or 14 oz."[2] The writer would advise operators to be content with the smaller amount in most cases, unless the rectum be extremely capacious or it be desirable, in case of a large stone, to give extra elevation and steadiness to the bladder. Even after distension of the bag with 12 oz. thrown in steadily and gently, a little blood-stained mucus followed its withdrawal at the close of the operation. No subsequent trouble followed, but it is evident that in injection of larger amounts some risk is run of damaging the rectal mucous membrane.

Injection of the bladder.—By means of a full-sized, soft catheter, an india-rubber bottle or a good-sized syringe

[1] 'Annales des Maladies des Organes Genito-urinaires,' Tom. i

[2] M. Guyon, loc supr. cit., gives 450 to 500 cc., or 15¾ oz. to 17½ oz.

8 or 10 oz.[1] of some antiseptic fluid are gently thrown in. By this double distension of rectum and bladder the latter will probably be both seen and felt reaching two thirds of the way between the umbilicus and pubes. The catheter should now be withdrawn from the bladder and a Jaques' india-rubber catheter tied round the penis. If the bladder does not seem to be sufficiently prominent a little more fluid may be thrown into the rectal bag and into the bladder.

The writer would conclude with the following propositions:

1. That supra-pubic lithotomy, as recently modified, has a future of revived usefulness before it, and that while, as an operation, it can never contrast with the rapid brilliancy of the lateral operation, it will be found of great value by those who only have to deal with stone occasionally, and who find themselves face to face with calculi of considerable size in adults.

2. That, to give other and more individual instances, the operation will be found useful in (*a*) many cases of hard stones of one and a half inches in diameter; (*b*) in multiple hard stones; (*c*) in cases of calculus not phosphatic, occurring with enlarged prostate; (*d*) in some cases of foreign body in the female bladder with abundant calculous deposit (Sir H. Thompson).[2]

In the rarer cases of (*e*) a state of urethra which will not admit of the use of a lithotrite; (*f*) in a very deep perineum; (*g*) in a child with deformed pelvic outlet; (*h*) in a patient with ankylosed hip-joint not admitting of his being placed in the usual lateral lithotomy position (Sir H. Thompson).[3]

3. That at present, till a larger number of cases of the improved operation have been collected, it will be wiser not to attempt to close the bladder with sutures.

[1] M. Guyon, loc. supr. cit. gives 250 to 300 cc., or 8¾ oz. to 10½ oz. These amounts given here and in a preceding note for bladder and rectum correspond to those of Dr. Fehleisen (Berlin), 'Arch. für klin. Chir.,' Bd. xxxii, Hft. iii.

[2] Loc. supr. cit., p. 12.

[3] Loc. supr. cit.

4. That in reviving an abandoned operation these two questions call for an answer:

A. Do we stand in a better position towards the operation than did our predecessors?

This question can only be answered in the affirmative after the work done by Dr. Garson, Prof. Petersen, and Sir H. Thompson.

B. On what grounds was the operation abandoned? The chief of these appear to have been: (1) The absence of any means of certainly avoiding the peritoneum. (2) The difficulty of sufficiently and painlessly distending the bladder in pre-anæsthetic days. (3) The absence of antiseptic fluids. (4) The fact that the operation was usually reserved for very large stones, and that it was often performed for such stones after lateral lithotomy had been recently attempted either on the same or the preceding day.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' New Series, vol. ii, p. 94.)

THE CHEMICAL PATHOLOGY

OF

RESPIRATION IN CHOLERA.

BY

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It has been often asserted, and even still more often assumed, that cyanosis is not only distinctive of choleraic collapse, but that it is due to an excess of carbonic acid in the blood; and many useless, if not injurious, attempts have in consequence been made to increase the amount of oxygen in the blood of the pulmonary veins by the inhalation of hyperoxygenated air during the stage of collapse. The exceptional importance which has been ascribed to cyanosis in relation to cholera has not only led to much error both as regards diagnosis and treatment, but also to a widespread and an almost unquestioning belief that the disease is essentially associated with defective oxygenation of the blood. As regards diagnosis it will be sufficient for me to refer to one of my papers "On some Physiological Errors connected with Cholera,"[1] in which

[1] 'Lancet,' Nov. 11th, 1871, pp. 670, 671.

it has been shown that cyanosis is liable to occur during the collapse resulting from rapidly fatal poisoning by croton oil, by arsenic, by corrosive sublimate, and other preparations of mercury; by the mineral acids; from perforation of the stomach; and from obstruction, strangulation, rupture, and perforation of the small intestines. In such cases, which often closely simulate cholera, it has been observed that the skin is not unfrequently cyanosed, and is sometimes "even more blue than is usual in cases of true cholera." This occurrence of cyanosis in connection with gastro-intestinal affections had been fully recognised early in the present century by Broussais; and later and special writers on the subject have appropriately grouped some of these cases together under the heading of "gastro-intestinal cyanosis."[1]

In the above-quoted paper, "On some Physiological Errors connected with Cholera," attention was particularly directed to the fact that "there is a local disappearance of cyanosis during choleraic collapse, when galvanism is applied to a limb, which is independent of any effect produced on the pulmonary circulation." This influence of galvanism on the cyanosis of cholera is in no respect exceptional, for it will be found on referring to the joint report of Drs. Russell and Barry, dated "St. Petersburg, $\frac{27}{15}$ July, 1831," that, on the first introduction of the disease into Europe, it had been observed that "frictions remove the blue colour for a time from the part rubbed." The effect produced on these occasions by galvanism and by frictions evidently cannot be ascribed to any consequent increase of oxygen in the blood, for the only internal respiratory change which could result from thus urging on the blood through the tissues, would be a local increase of oxidation; as the oxygen already present in the blood would by such means be more quickly withdrawn from it

[1] Cited by F. G. Boisseau, "Mémoire sur la Cyanose Cholérique," 'Journal Univ. et Hebdom. de Méd. et de Chir. Prat.,' 1832, tome ix, pp. 277—298. See also F. J. V. Broussais, 'Le Choléra-Morbus Epidémique, observé et traité selon la Méthode Physiologique,' 1832, pp. 75, 76.

and used in that process of tissue change which is represented by an increased formation of carbonic acid. Before concluding these preliminary remarks it will be useful to direct attention to the fact, that even the normal change of colour from red to dark, which is chiefly effected in the capillary circulation, cannot be physiologically assigned to the larger quantity of carbonic acid which venous blood contains; since it has been shown by Pflüger, that "if equal quantities of oxygen be added to two portions of blood, and if carbonic acid be added to one of them, the colour is not changed."[1] Consequently, as the cyanosis of cholera fails to indicate the extent to which the aeration of the blood either has or has not been performed, it becomes necessary to analyse the air expired during life, and to examine the lungs after death in order to prove whether there is or is not any connection between this so-called asphyxia and choleraic collapse.

Those who are familiar with the literature of cholera know that it is very rich in evidence which proves that during the stage of collapse the respiration is usually much diminished, and that after death, in the case of those who die before reaction has begun, the lungs are always more or less collapsed. But when, in addition to such evidence, attention is directed to the numerous and exact analyses which have been made of the expired air, it will be found that the net result of the pulmonary interchange of gases in this disease has always been a relatively large gain in the amount of oxygen received by the blood in exchange for carbonic acid, as compared with that which is relatively gained by such interchange of gases during health. When referring to the works of those observers who have specially devoted their attention and skill to this subject, a decided preference should be given to M. Doyère's 'Mémoire sur la Respiration et la Chaleur Humaine dans le Choléra' (1863), as it is founded

[1] Cited by Landois, 'A Text-Book of Human Physiology,' translated from the fourth German edition. With additions by William Stirling, M.D., Sc.D., vol. i, 1885, p. 60.

on a very large number (nearly 300)[1] of careful observations made in 1849; as the results then obtained were confirmed by a second series of observations made in 1854, under the direction of a committee appointed for that purpose by the French Academy of Sciences, but of which the literary results were unfortunately lost; and, lastly, as the great value of the work was authoritatively recognised in 1858 by a subsequent committee, composed of MM. Serres, Velpeau, Cl. Bernard, Jules Cloquet, Jobert de Lamballe, and Andral, and whose report in its favour led to a prize of 5000 francs from the Bréant foundation being awarded to M. Doyère early in the following year (March 14th, 1859). Previous to the dates of these researches it had been analytically proved in 1819 by Dr. John Davy and Mr. Finlayson,[2] during an epidemic of cholera in Ceylon, that the air expired during choleraic collapse is "very deficient in carbonic acid;" the amount of carbonic acid, as compared at the time with that expired

[1] 'Comptes-rendus Hebd. des Séances de l'Acad. des Sciences,' October 22nd, 1849, p. 454.

[2] The importance of investigating the composition of the air expired by cholera patients was first recognised by Dr. John Davy at the latter end of April, 1819; and it was soon after this date that he had the opportunity of personally communicating his ideas on the subject to his "very intelligent and worthy friend, Mr. Finlayson," whose early death was due to phthisis, which was contracted during the following year in Siam. The first analysis of the air expired by a cholera patient was made jointly by these observers, and Dr. Davy states that "at my desire, Mr. Finlayson was so good as to continue the inquiry at a time I had no opportunity of continuing it myself." The results of these analyses were communicated to Dr. Davy in a letter dated "Kandy, 4th June (1819)." 'Report on Cholera, as it occurred in Ceylon in 1819' (published from a copy in the author's possession), by John Davy, M.D., F.R.S., &c.; 'Medical Times,' Aug. 31st, 1850, pp. 224—226; and also in his work 'On some of the more important Diseases of the Army, with contributions to Pathology,' 1862, pp. 113—122. Although this report remained unpublished for considerably more than thirty years after its transmission from Ceylon to the Medical Board of the Army, yet it was not immediately shelved; for Sir Gilbert Blane had the opportunity of reading it in manuscript soon after its arrival in this country, and he gave a summary of its contents at a meeting of the Medical and Chirurgical Society on June 6th, 1820 ('Med.-Chir. Trans.,' vol. xi, 1820, pp. 157—164).

by a healthy person of the same country and race, having been found in the first case to be only one fifth; in the second case one third; and in the several other cases examined, to be much below the normal standard. Whilst M. Rayer,[1] physician to "la Charité" Hospital, Paris, analytically recognised, in 1832, that there is a diminished quantity of oxygen absorbed. But it was reserved for M. Doyère to prove that in addition to these important but detached facts, which simply indicate a great reduction in the interchange of gases in the lungs, that there is a relatively large amount of oxygen absorbed, which, as regards the respiratory quotient of health, is constantly and sometimes very greatly in excess of that which can be accounted for by the carbonic acid eliminated.[2] Since the date of M. Doyère's researches there have been other, and some improved, methods for ascertaining the relative amounts of oxygen absorbed and of carbonic acid eliminated, both as regards health and disease; in consequence of which the respiratory quotient of health, according to the best authorities of the present day, $\frac{CO_2}{O}\left(=\frac{4{\cdot}38}{4{\cdot}782}\right)=0{\cdot}906$,[3] expresses a larger proportion of oxygen than that given, as the mean of twenty-one analyses, by M. Doyère in 1849, $\frac{CO_2}{O}\left(=\frac{4{\cdot}36}{4{\cdot}47}\right)=0{\cdot}977$.[4] But this does not affect the

[1] "Examen comparatif de l'air expiré par des Hommes Sains et des Cholériques, sous le rapport de l'oxygène absorbé," 'Gazette Médicale de Paris,' 26 Mai, 1832, pp. 277, 278.

[2] Notwithstanding this relatively large excess of oxygen aborbed, it was assumed by M. Doyère, in his introductory remarks, that asphyxia is "the constant phenomenon of cholera." The chief evidence, according to M. Doyère, in favour of asphyxia, is "the diminution of the proportion of carbonic acid produced and of oxygen absorbed;" and he proceeds to add, in accordance with the prevailing opinion of his day, that "the symptom most intimately associated with choleraic asphyxia is, I have hardly need to say, cyanosis."

[3] Dr. F. Landois, op. cit., p. 225.

[4] M. Doyère's observations in 1849, on the average amount of oxygen absorbed during healthy respiration, 4·47 per cent., agree very closely with those of M. Rayer in 1832, who found the mean of thirteen analyses to be 4·45 per cent.

general results of his researches as regards the relatively larger amount of oxygen absorbed in proportion to the carbonic acid eliminated during cholera, as compared with the relation between these two gases observed by him in the air expired during health. In the case (No. 6), for example, of a lad, æt. 16, who was admitted into the Hôtel Dieu, Paris, at 4 p.m., on April 28th, 1849, in a state of "extreme algidity," with strongly marked cyanosis and suppression of urine since the previous evening, the analysis of the air expired thirty minutes after admission showed that there was a reduction in the interchange of gases in the lungs to considerably less than half of the normal amount; and, at the same time, a relatively large excess of oxygen absorbed in proportion to the amount of carbonic acid eliminated. It was moreover observed during the progress of this case, in which, between April 28th and May 7th, fourteen observations were made on the composition of the expired air, that there was a relative excess of oxygen, associated with an absolute reduction in the pulmonary interchange of gases, both during reaction as well as during collapse.

This important fact in the chemistry of respiration in cholera shows that the blood which is conveyed to the lungs by the pulmonary arteries becomes relatively more oxygenated during its passage onwards to the pulmonary veins than is the case during health; and it has been fully established by numerous and trustworthy analyses of the air expired during cholera, that however low the absolute amount of oxygen absorbed may fall during the pulmonary interchange of gases, it is always relatively, and sometimes very largely, in excess of the amount of carbonic acid eliminated. For it has been clearly demonstrated that the blood which is brought to the lungs during choleraic collapse for the purpose of aeration, gives up a relatively diminished amount of carbonic acid in return for the oxygen taken in, owing to the formation of carbonic acid in the system having been greatly reduced, and that consequently when it leaves the lungs by the pulmonary

veins, it is relatively far richer in oxygen than is normally the case. This has been satisfactorily illustrated in the following case (No. 31), observed by M. Doyère, of a journalist, æt. 33, who was admitted into the Hôtel Dieu on May 24th, 1849 at 2.30 p.m., and who died, during choleraic collapse, at 9.15 p.m. on the same day. In this typical and rapidly fatal case of cholera there was, throughout the progress of the disease, a relatively large excess of oxygen absorbed in comparison with the amount of carbonic acid eliminated, as is well shown in the following series of analyses of the expired air. At 3 p.m., or thirty minutes after the patient's admission, the respiratory quotient was found to be $\frac{CO_2}{O}\left(=\frac{1{\cdot}61}{2{\cdot}23}\right)$ $= 0{\cdot}72$; at 4 p.m., $\frac{CO_2}{O}\left(=\frac{1{\cdot}71}{2{\cdot}44}\right)= 0{\cdot}70$; at 4·45 p.m., $\frac{CO_2}{O}$ $\left(=\frac{1{\cdot}62}{2{\cdot}32}\right)= 0{\cdot}70$; and at 5.25 p.m., $\frac{CO_2}{O}\left(=\frac{1{\cdot}57}{2{\cdot}15}\right)= 0{\cdot}73$. The average quantity of carbonic acid eliminated from the lungs in this case, according to these four analyses, was consequently reduced to 37 per cent., whilst the oxygen absorbed was only a fraction below 54 per cent. of the normal amount. When the concluding observation of the air expired in this case was made at 8.5 p.m., and when the temperature in the armpit was 37·8° C., there was found, as the mean of three analyses, to be a very much greater disproportion between the amount of oxygen absorbed and the amount of carbonic acid eliminated, for the respiratory quotient was then only $\frac{CO_2}{O}\left(=\frac{\cdot 23}{1{\cdot}30}\right)=0{\cdot}18$. Consequently at the time of this last observation, which was made one hour and ten minutes before death, the carbonic acid eliminated from the lungs was not more than about 5¼ per cent., whilst the oxygen absorbed was 29 per cent. of the normal amount; or, in other words, the oxygen absorbed was equal to nearly six times the carbonic acid eliminated. A correspondingly large excess of oxygen absorbed shortly before death was also

very noticeable in other cases, and especially in that of a wood-sawyer æt. 37 (No. 12), who died during collapse sixteen hours after the commencement of the disease. Five minutes before death, and when the temperature of the armpit had risen to 38·3° C., the respiratory quotient, $\frac{CO_2}{O}\left(=\frac{\cdot 84}{2\cdot 10}\right)=0\cdot 40$, showed that whilst the carbonic acid eliminated was only 20 per cent., the oxygen absorbed was 47 per cent. of the normal amount.

There is no evidence, derived from the chemistry of respiration in cholera, in favour of the supposition that in well-marked and typical cases of the disease, carbonic acid is either accumulated in the system during collapse, or that there is an exceptional excess of it in the venous blood waiting, as it were, to escape through the lungs as soon as reaction should occur. On the contrary, it has been observed that in the same way that the first urine passed after its previously more or less prolonged suppression is deficient in urea, so the air expired during well-marked reaction is correspondingly deficient in carbonic acid; and such deficiency is observable both in those cases in which reaction ends in death, as well as in those cases in which recovery occurs ofter a more or less prolonged and well-marked stage of convalescence. In the case (No. 2) of a young man, æt. 24, who was admitted into the Salpêtrière Hospital, Paris, on April 17th, 1849, with well-marked reaction consequent on a very severe algide stage of the disease, the pulse was 70 and fairly good, and there were only 20 to 22 very natural inspirations per minute, although the cyanosis was still very pronounced. The analysis of the air expired in this case, soon after admission, showed that the carbonic acid eliminated was only half of the normal quantity, whilst the oxygen absorbed was relatively in great excess, the respiratory quotient being $\frac{CO_2}{O}\left(=\frac{2\cdot 10}{2\cdot 78}\right)=0\cdot 75$. On April 20th, about thirty-four hours previous to death, and when the patient had been in a very grave typhoid state since the previous day, it was found,

on analysis, that the carbonic acid eliminated was reduced to one third of the normal quantity, whilst the oxygen absorbed was relatively in almost the same degree of excess as in the preceding analysis, the respiratory quotient being $\frac{CO_2}{O}\left(=\frac{1{\cdot}42}{1{\cdot}92}\right)=\cdot74$. In like manner, when the stage of reaction is followed by recovery, there is a corresponding reduction, as regards the interchange of gases, with a relatively more or less considerable amount of oxygen absorbed, as occurred in the preceding case, in which death occurred during reaction. This has been well illustrated in the case (No. 3) of a woman, æt. 30, who was admitted into the same hospital and on the same day as the last cited case; and who, at the time of her admission, was in the stage of commencing but very decided reaction, with 28 inspirations per minute, and with a slight return of the urinary secretion. The analysis of the air expired in this case, soon after admission, showed that the carbonic acid eliminated was only half of the normal quantity, whilst the oxygen absorbed was relatively in decided excess, the respiratory quotient being $\frac{CO_2}{O}\left(=\frac{2{\cdot}17}{2{\cdot}47}\right)=0{\cdot}88$. Three days later on, when reaction had been succeeded by convalescence, and the urinary secretion had been completely restored, the carbonic acid eliminated was still barely more than half of the normal quantity, whilst there was relatively a large excess of oxygen absorbed, the respiratory quotient being $\frac{CO_2}{O}\left(=\frac{2{\cdot}34}{2{\cdot}96}\right)=0{\cdot}79$.

These observations on the chemistry of respiration in cholera, and especially as regards the period of reaction, are strictly in accordance with the thermometric observations of MM. Briquet and Mignot, and of other recognised authorities on the subject. From the carefully tabulated observations of MM. Briquet and Mignot[1] on eighty-six patients suffering from the disease, it appears that although the period of reaction is usually accompanied by a compara-

[1] 'Traité Pratique et Analytique du Choléra-Morbus,' 1850, pp. 299, 300.

tively small elevation of temperature, which "at the most is not more than 2° to 3° Cent., more often 1°, and even only some tenths of a degree;" yet some of their observations have served to show that "there exists, not only during the algide period, but even during all the continuance of the choleraic phenomena, a tendency to coldness, in virtue of which the reduction of temperature is in some cases more pronounced at the period of reaction than in the cyanic period."

If any further evidence were needed to prove that cholera is unconnected with defective oxygenation of the blood, it would be unnecessary to do more than refer to that afforded by the pulmonary interchange of gases when the urinary secretion has been restored. For it has been clearly demonstrated that whilst the previously prolonged suppression of urine has always coincided with a great reduction in the amount of carbonic acid eliminated, and with a relative excess in the amount of oxygen absorbed, the restoration of the urinary secretion is not preceded, nor even for some days necessarily followed, by any corresponding difference in the interchange of gases in the lungs. In the case (No. 6) already cited, of a lad, æt. 16, in which the urine was completely suppressed from the evening of April 27th to the evening of April 29th, the lowest respiratory quotient during the intervening time was found to be $\frac{CO_2}{O}\left(=\frac{1{\cdot}58}{1{\cdot}92}\right) = 0{\cdot}82$; showing that whilst the carbonic acid eliminated was only 36¼ per cent., the oxygen absorbed was 43 per cent. of the normal amount. When the urinary secretion in this case had been restored about twelve hours (April 30th, 9 a.m.), the respiratory quotient $\frac{CO_2}{O}\left(=\frac{1{\cdot}95}{2{\cdot}50}\right) = 0{\cdot}78$, showed that the carbonic acid eliminated was 45 per cent., and the oxygen absorbed was 56 per cent. of the normal amount. Three days later on, May 3rd, 9 a.m., when the urine had become abundant, the respiratory quotient, $\frac{CO_2}{O}\left(=\frac{2{\cdot}09}{2{\cdot}46}\right) = 0{\cdot}85$, showed

that the carbonic acid eliminated was 48 per cent., and the oxygen absorbed was 55 per cent. of the normal amount. Finally, on May 7th, at 5.30 p.m., when the last analysis was made, the respiratory quotient, $\frac{CO_2}{O}\left(=\frac{2{\cdot}72}{2{\cdot}99}\right) = 0{\cdot}92$, showed that the carbonic acid eliminated was 62½ per cent., and the oxygen absorbed was 67 per cent. of the normal amount. It will be sufficient to add that in cases like this, which is typical of what occurs both during and subsequent to choleraic collapse, neither the previously prolonged suppression, nor the succeeding abundance, of the urinary secretion could have been influenced by any variations in the interchange of gases in the lungs; for during the ten days that the case was under special observation, the relative and continued excess of oxygen absorbed was limited to the comparatively narrow range of 4½ to 11 per cent. above the standard proportion of health.

The chemistry of respiration during the stage of choleraic convalescence has been as yet very imperfectly studied. But there is some evidence to show that the tendency to excess in functional activity, which, as regards the renal secretion, leads to temporary glycosuria, may also lead, as regards the pulmonary function, to an absorption of oxygen which may, for a comparatively short time, be absolutely greater than the standard of health. In one of M. Doyère's cases (No. 7) it was noted, fourteen days after the commencement of the disease, when the pulse was 64 per minute, and the health appeared to be "perfectly re-established," that the respiratory quotient was $\frac{CO_2}{O}\left(=\frac{3{\cdot}40}{4{\cdot}98}\right) = 0{\cdot}68$; showing that the carbonic acid eliminated was still only 78 per cent., whilst there was an absolute excess of oxygen absorbed to the extent of 11½ per cent. above the normal standard. In two other cases (Nos. 8 and 14) moderate reaction from slight collapse was observed to lead to an absolute excess in the absorption of oxygen, which, in each case, was also above, although only to a small extent, the normal standard

(analyses 43 and 66). Whilst in a fourth case (No. 38) it was observed during a convalescent period of five days, extending from the eighteenth to the twenty-third day after admission into the hospital, when the average temperature of the armpit was 37° C., and the average pulse was 57 per minute, that the amount of oxygen absorbed, although not quite up to the normal standard, was relatively very large; for the respiratory quotients, $\frac{CO_2}{O}\left(=\frac{3{\cdot}40}{4{\cdot}40}\right)=0{\cdot}75$, $\frac{CO_2}{O}\left(=\frac{3{\cdot}57}{4{\cdot}27}\right)=0{\cdot}83$, $\frac{CO_2}{O}\left(=\frac{3{\cdot}55}{4{\cdot}29}\right)=0{\cdot}83$, and $\frac{CO_2}{O}\left(=\frac{3{\cdot}39}{4{\cdot}14}\right)=0{\cdot}82$, showed that the average amount of carbonic acid eliminated was still below 80 per cent., when that of the oxygen absorbed was 96 per cent. of the normal standard. It is important also to note in this last case that during the succeeding eleven days which the patient continued to pass under special observation, when the average pulse was 63 per minute, and the average temperature was 37·4° C., there was a relative excess instead of a relative deficiency in the amount of carbonic acid eliminated; and at the same time loss of appetite instead of the previous desire for food. These observations on the chemistry of respiration during choleraic convalescence, like those on the occurrence of temporary glycosuria as a sequel to cholera,[1] show that "the tendency to excess during recovery from a central arrest of nutrition" does not readily cease.

There are some physiological facts connected with the chemistry of respiration in health which may with advantage be referred to in connection with the chemistry of respiration in this disease. It will be sufficient, however, for me on this occasion to state that the quantity of oxygen absorbed in the lungs is only to a very small, if any, extent influenced by an artificially produced excess of oxygen in the air for inhalation; and that if the deficiency of carbonic acid in the air expired by cholera patients during collapse, and to a less extent during convalescence, be

[1] 'Medico-Chirurgical Transactions,' vol. liv, 1871, pp. 63—93.

considered in connection with this as well as with other and allied physiological facts,[1] there will be less difficulty in understanding why such deficiency cannot be referred to any unsatisfied demand of the blood for oxygen. For whilst the analysis of the expired air demonstrates that the net result of the pulmonary interchange of gases is relatively very favorable as regards a clear gain of oxygen, all attempts to still further oxygenate the blood by the inhalation of additional supplies of oxygen have signally failed during each successive outbreak of the disease. Somewhat more than fifty-four years have passed since it was recorded by Dr. W. B. O'Shaughnessy,[2] whose name was at one time well known in connection with the chemical pathology of cholera, "that the inhalation of oxygen gas has failed remarkably in achieving the desired end is unhappily too notorious." This failure, it may be added, has not been due to any difficulty as regards inhalation, but simply to the absence of any demand on the part of the coloured blood-corpuscles for additional supplies of oxygen beyond what is contained in atmospheric air. For it has been very clearly shown that the great and remarkable affinity for atmospheric oxygen, which physiologically characterises the coloured blood-corpuscles, or rather the hæmoglobin which constitutes more than nine tenths of their bulk, instead of being lessened is increased in this disease.

[1] (*a*) That the amount of oxygen normally present in arterial blood is barely more than half the amount of carbonic acid; the proportion being 17 volumes of oxygen to 30 volumes of carbonic acid in 100 volumes of such blood.

(*b*) That the blood, in becoming venous, does not gain more per cent. than from 5 to 7 volumes of carbonic acid, whilst it loses from 8 to 12 volumes of oxygen; and that consequently the oxygen absorbed during the subsequent aeration of the blood in the lungs, is normally in excess of the carbonic acid eliminated.

(*c*) That during hybernation, when the pulmonary interchange of gases is extremely reduced, the oxygen absorbed ($\frac{1}{11}$) is almost double the amount ($\frac{1}{16}$) of the carbonic acid eliminated.

[2] "Proposal of a New Method of Treating the Blue Epidemic Cholera by the Injection of highly-oxygenated Salts into the Venous System," 'Lancet,' Dec. 10th, 1831, p. 367.

In thus attempting to recapitulate, as concisely as possible, some of the more important observations which have been made and recorded in connection with the chemistry of respiration in cholera, attention must be chiefly directed to the fact that whilst the absolute amount of interchange of gases in the lungs is always much reduced, in consequence of the formation of carbonic acid in the system having been partially arrested, that there is in this disease, and more especially during its stage of collapse, a relatively large amount of oxygen absorbed, which, as regards the amount of carbonic acid eliminated, is usually much above the standard proportion of health. This relative excess of oxygen absorbed necessarily leads to an almost exhaustive elimination of carbonic acid from the lungs, and to the blood, in its passage onwards to the pulmonary veins, becoming, as already stated, surcharged with oxygen. The great reduction in the supply of carbonic acid to the lungs, which is strictly in accordance with the continued ability of the patient, even during profound collapse, to make a moderately full inspiration, and also with the comparatively favorable character of the auscultatory signs of respiration, which indicate that there is no obstruction to the entrance of air, appears to be essentially connected with each stage of the disease. One of the earliest changes affecting the respiratory movements in cholera, and which is primarily due to this deficiency in the supply of carbonic acid to the lungs, is the ineffectual prolongation of the inspiratory murmur, and the exceptional shortening of the expiratory murmur, which lead to diminution, and ultimately to more or less complete failure of the voice. The duration of the inspiratory murmur has been observed, in a large number of cases of cholera, to be about twice as long as the expiratory murmur, during prolonged and well-marked collapse. In one of the cases specially noted by the late Dr. Parkes,[1] the relation between the two was as 12 to 5; in another

[1] 'Researches into the Pathology and Treatment of the Asiatic or Algide Cholera,' 1847, p. 67.

case, as 6 to 4; and in a third case it was twice as long; whilst the respiratory rhythm of health is as 6 to 7 or 8. This failure of the voice has been very commonly spoken of as the vox cholerica, but it is decidedly incorrect to refer to it as a diagnostic sign of choleraic collapse; for a corresponding failure of the voice, amounting in some cases to complete aphonia, has been noted by myself and by other observers in gastro-intestinal cases, in which there has been collapse simulating that of cholera. In such cases, as in cholera, there is a well-marked and characteristic change in the respiratory function during life, and, not unfrequently, a collapsed state of the lungs after death, which must be ascribed to a diminished supply of carbonic acid to the lungs, consequent on a previously diminished formation of carbonic acid in the system.

This failure from reduced production of carbonic acid, combined with the relative excess of oxygen absorbed, is moreover in accordance with the very decided influence of cholera on the dyspnœa of phthisis, which has for a long time attracted much attention; owing to the pathological effect of phthisis on the lung, as an organ for the elimination of carbonic acid, being necessarily to reduce its efficiency. For it has been carefully noted by MM. Briquet and Mignot[1] who, in common with other trustworthy observers, have had favorable opportunities for observing the not unfrequent occurrence of cholera in conjunction with this disease, that "in all our phthisical patients we have constantly seen the dyspnœa diminish, and the expectoration nearly or completely cease."

The physical signs of respiration and the analysis of the expired air show that the much reduced amount of blood supplied to the lungs continues to be well oxygenated during choleraic collapse. But it is chiefly by means of exact examinations after death of the extremely contracted lungs themselves, in those cases in which death has occurred before any reaction has commenced, that the extent to which carbonic acid has been eliminated during

[1] Op. cit., 1850, p. 360.

life can be fully estimated. With regard to the condition of the lungs after death, it should be noted that when attention was first directed to their contracted appearance in these cases, it was somewhat hastily, but not perhaps very unreasonably, assumed by some observers, that their condition must be due to the presence of air in the pleural cavities, which was thought to be alone capable of so completely overcoming the atmospheric pressure. At an early period in the first great epidemic of the disease in the Madras Presidency, an able observer, Dr. Pollock, of H.M's. 53rd Regiment, availed himself of an opportunity for opening, within two hours after death, the thorax of the dead body of a cholera patient under water; and as no gas was extricated, it became evident that the contracted condition of the lungs was not due to this, but to some intra-pulmonic cause.[1] Before however, any other suggestion on the subject could be reasonably offered, it obviously became important to demonstrate the exact nature as well as the extent of the pulmonary collapse; and this work has been satisfactorily done by the late Dr. Parkes,[2] whose researches have been fully confirmed by Dr. Sutton, by myself, and by very many other observers. Dr. Parkes has demonstrated that the lungs in these cases are less crepitant than usual, and that their specific gravity is diminished; showing that there is not only absence of air, but also of blood. The extent of the pulmonary collapse was found to be very considerable; for of thirty-nine cases in which the condition of the lungs was very carefully investigated by Dr. Parkes, it was ascertained that "in fourteen cases the lungs were completely collapsed, appearing in some cases almost like the lungs of a fœtus. In three cases they were considerably, and in eight cases they were slightly collapsed; and in the remaining fourteen cases, the collapse was in some cases altogether, and in other cases partially prevented by old

[1] Scot (W), 'Madras Report on Cholera,' 1824, p. 225, and Preface, p. xxxiii. See also Dr. Parkes, op. cit., 1847, p. 121.

[2] Op. cit., 1847, pp. 14—17.

adhesions." Dr. Parkes states, as the result of this collapsed condition, that "in twenty-four cases, the crepitation was totally abolished; in fifteen cases it was notably diminished in some part of the lung, and in one of these abolished completely in the upper lobes. The want of air was not owing to mechanical impediment, as on artificial respiration air passed readily in, distended the before collapsed lung, and partially or wholly restored the crepitation. This," Dr. Parkes proceeds to state, "I proved by many trials." Whilst the diminution of weight in the case of both lungs, consequent on reduced supply of blood, was found by Dr. Parkes to average 20 oz.; assuming the healthy standand weight for both lungs in males to be, according to Dr. Clendinning 46 oz.

The abolition of crepitation would thus appear to be both coextensive and coincident with the reduced supply of blood, and to be consequent on the smaller ramifications of the air-vessels having been gradually contracted so as to exclude the atmospheric air, at the same time that the previously reduced supply of carbonic acid has been more or less fully eliminated from the blood conveyed by the pulmonary arteries for aeration; and which passes onwards through the pulmonary veins, with a relative excess of oxygen to the left side of the heart. For whilst the relative excess of oxygen absorbed during health has the effect, so far as the pulmonary function is concerned, of assisting to promote the passage of blood through the lungs, the relatively larger excess of oxygen absorbed, during the collapse resulting from cholera and from allied conditions of the system, assists in still more effectually promoting the pulmonary circulation, which by this means is continued under great and increasing difficulties until the slowly diminishing supply of carbonated blood to the lungs almost or finally stops. The abolition of crepitation, like the diminished amount of blood, is in the same manner due simply to failure as regards both supply and demand; for although the well-known tendency to diffusion between the carbonic acid passing outwards from the air-

vesicles and the oxygen passing inwards from the bronchial tubes is relatively still unchecked, yet the chemical interchange of gases in the blood of the pulmonary capillaries steadily decreases with the advancing collapse, until, like the passage of the blood through the lungs, it slowly and completely fails. From the numerous observations which have been made on the progressively reduced frequency of breathing which immediately precedes death during choleraic collapse, it will be sufficient to select a fairly typical case reported by Dr. F. Paschall,[1] in which the respirations were specially timed "during the last five minutes of life, and were as follows: first minute 20; second 15; third 12; fourth 6; 5th 1 deep inspiration."

The resulting collapse of the lungs in such cases would therefore be due not to any morbidly excited contraction of the parietes of the smaller subdivisions of the pulmonary blood-vessels or of the air vessels, but to the natural elasticity of the lungs themselves, which specially favours the exclusion but not the entrance either of blood or of air, when the formation of carbonic acid in the system has been more or less extensively checked. From the thoroughly trustworthy observations of Dr. Parkes it is evident that as the lungs after death in some cases of cholera are so completely collapsed as to appear "almost like the lungs of a fœtus," the previous interchange of gases must have become less and less before it quite ceased; and that as the supply of blood sent to the lungs for aeration is to a great extent dependent on the amount of carbonic acid which it contains, this excretory product, which qualifies, as it were, the blood for aeration, must in like manner have been previously very much reduced before the pulmonary circulation could have so completely failed as to leave the lungs almost without blood as well as almost without air. The fact observed by Prof. Griesinger, that percussion during choleraic collapse gives a small area of cardiac dulness, shows that this failure in the supply of blood to the lungs is associated with a dimi-

[1] 'The Cholera Epidemic of 1873 in the United States,' 1875, pp. 18, 19.

nished amount of carbonated blood in the right cavities of the heart, and consequently in the pulmonary arteries, during life;[1] whilst the relative excess of oxygen, which is conveyed by the blood from the lungs to the left side of the heart, accounts not only for the remarkable integrity of the mental faculties during collapse, but also for the state of the left ventricle after death, which "is often found so firmly contracted that it must have closed forcibly on the last drops of blood that entered it."[2] The presence moreover of such relative excess of oxygen in arterial blood, thus stimulating into increased activity the vaso-motor centre, supplies a more satisfactory explanation of the emptiness of the brachial and other large arteries during advanced periods of collapse, which has been experimentally demonstrated by Magendie, Dieffenbach, and other observers, than the increased venosity of the blood, to which the general emptiness of the arteries after death has been very commonly referred. For this increased venosity of the blood, which occurs both shortly before as well as after death, is a capillary and not an arterial change; and it can therefore only have a secondary and an altogether indirect influence in contributing to any arterial expulsion of blood.

The not unfrequent association of collapse closely resembling that of cholera in cases such as those which have been referred to in my paper "On some Analogies of Cholera, in which Suppression of Urine is not accompanied by Symptoms of Uræmic Poisoning,"[3] with a similarly contracted condition of the lungs after death, shows that such pulmonary contraction is not only independent of any cause which is peculiar to cholera, but that it is necessary to seek elsewhere than in the lungs themselves for the primary change which has led to this result; and, in thus following analogy as a guide, we may not unreasonably expect that it will lead us to recog-

[1] Cited by Mr. Simon, 'Ninth Report,' 1866, p. 429, note.
[2] Dr. Parkes, op. cit., 1847, pp. 105, 106.
[3] 'Med.-Chir. Trans.,' vol. li, 1868.

nise that in the same way that the non-appearance of urine in the bladder is due to deficiency and arrest of urea formation in the system, and is independent, at least to a very great extent, of the kidneys; so, in like manner, the reduced interchange of gases and subsequent condition of pulmonary collapse are due to a corresponding deficiency and partial arrest of carbonic acid formation in the system, and are independent of any morbid condition of the lungs themselves. The greatly reduced but continued formation of carbonic acid during collapse, when that of urea has been thus almost if not completely stopped, is undoubtedly due to carbonic acid being a lower compound than urea, which, from a more or less strictly chemical point of view, might conveniently be referred to as a diamide of carbonic acid, or simply as a carbamide; and if, in accordance with recent progress in chemical science, we adopt one of these newer titles for urea, it would perhaps be more easy to recognise why, during choleraic collapse, the formation on a greatly reduced scale of carbonic acid in the tissues, or possibly in the blood itself, should continue, and the formation of a diamide of carbonic acid should cease.

It is perhaps almost unnecessary to add that the above cited facts connected with the chemistry of respiration in cholera do not admit of being otherwise explained. The great function of respiration is secured by being made to depend on simple and physical conditions, and it is therefore comparatively safe from such destructive influence of disease as is able in cholera to wreck the functions of those organs which are associated with nutrition, and which are affected, not by physical, but by peculiarly vital operations. This essential distinction between the function of the lungs on the one side, and the functions, for example, of the liver and the kidneys on the other, becomes still more noticeable when we pass from the consideration of the physically secured function of respiration, and from the vitally insecure and consequently wrecked functions connected with nutrition, to the rela-

tive influence of cholera on those structures and organs which are either directly or indirectly associated with reproduction. As this part of the subject has been already somewhat fully illustrated in my paper "On the Continuance of the Mammary Secretion during Collapse,"[1] it will be sufficient to state that the relative exemption there referred to is not limited to cases of this disease, but that it has been carefully noted in other cases in which there has been a central arrest of nutrition, and in which consequently the collapse has simulated that of cholera; as, for example, in acute poisoning by sulphuric acid.[2]

There remain to be noticed, and that very briefly, the great reduction of animal heat during collapse, and the remarkable increase of temperature shortly before death, which are both in accordance with the facts elicited by the chemical investigation of the respiratory function during life, and with the comparatively exsanguine and non-crepitant state of the lungs observed after death. As regards more especially the rise of temperature, which has been often recognised not only immediately before, but also after death, it is, as the result, at least to a very great extent, of temporarily increased oxidation, evidently dependent on a previous accumulation of oxygen. For it has been shown, by repeated analyses, that oxygen is continuously admitted into the system and to a great extent unconsumed during collapse; and therefore it would be ready to be thus used when life was becoming or had become extinct, and when consequently physical change was either ceasing or had ceased to be any longer checked by vital influence.

[1] 'British Medical Journal,' Sept. 19th, 1868.

[2] Casper, 'A Handbook of the Practice of Forensic Medicine, based upon Personal Experience,' translated from the third edition of the original by George William Balfour, M.D.St. Andrews, vol. ii, 1862, pp. 83, 84.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' New Series, vol. ii, p. 102.)

TWO CASES OF SPLENECTOMY.

BY

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On April 16th, 1884, E. R—, æt. 19, single, was admitted under my care at the Samaritan Hospital on the recommendation of Dr. McRitchie, of Huntingdon.

Condition.—Anæmic, but not emaciated, tongue covered with creamy fur, papillæ prominent, appetite good, does not suffer from flatulence, bowels confined, lungs, heart and kidneys healthy. Left side of abdomen distended by a smooth kidney-shaped fluctuant tumour which is dull all over its surface and is not overlapped by intestine. This tumour is very mobile. When the patient is at rest on her back, its upper part extends about two inches above and to the left of the umbilicus, and its lower part occupies the whole of the left side of the abdomen, and extends below the umbilicus well into the right iliac region. It can be pushed up under the ribs of the left side, so that its lower border is only slightly below the navel; this position causes pain and a dragging sensation far back in the left side of the abdomen.

Family history.—Unimportant.

History.—Has never had any serious illness. Two years

back had an attack of pain in the lower abdomen and was examined by Dr. Walker, of Peterborough, who discovered a swelling just to the left of the navel. Nine months later had a succession of severe attacks of pain, accompanied by difficulty in micturition. Has been steadily failing in health since, but has had no return of severe pain, and no further difficulty in micturating. For the last three months the swelling has occupied the lower abdomen, and has been steadily increasing in size. It sometimes moves up higher and then gives her the same dragging pain which she complains of when it is pushed up.

Menstruation began at fifteen, and was regular and painless, but for about a year the periods have been very scanty and the intervals prolonged; the last period is over about ten days and came on after an interval of seven weeks.

There was no tumour to be felt in the pelvis, and no evidence that the uterus was connected with the abdominal swelling.

Diagnosis.—I was in doubt as to the nature of the tumour, thinking that it might possibly be a dermoid ovarian with a long pedicle, but rather inclining to the view that it was a cystic kidney, though there were no distinct evidences of renal disease of any kind. I did not think it was the spleen because I could not feel the notch, and it seemed to me altogether too low in the abdomen.

On April 22nd I explored the abdomen by the ordinary median incision. When the tumour was exposed I at first thought that it was the left kidney, as the exposed part had not the colour of any splenic tumour I had seen, but on passing in my hand I found the left kidney in its proper situation, but could not find the spleen, and on extending the incision upwards it was evident that it was a cystic spleen. The omentum was adherent to the lower part of the tumour and this had been the cause of the dragging pain when the tumour was pushed up. I separated the omentum and ligatured its torn surface in two parts by transfixion. The lower part of the tumour was so thin

that a dark fluid with scales of cholesterine could be distinctly seen through its walls, and at one part there was a small protrusion about as large as a filbert which appeared to be a fluid hernia. Having turned the lower part of the tumour out of the abdomen, I grasped its pedicle between my left thumb and forefinger and transfixed it with a No. 3 Chinese silk ligature. Having tied it in two portions with these locked ligatures I passed another separate one round the whole pedicle, tied it, and cut them all short. On drawing down the pedicle to divide it the patient became cyanotic and so alarmingly collapsed that the pillows were removed from under her head and brandy was injected subcutaneously. She did not revive, and in order to cover up the abdomen, I cut the tumour away and took the strain off the pedicle, when she at once improved and I proceeded with the operation. No blood was lost during the ligature of the pedicle and separation of the tumour. There was very little sponging of peritoneum necessary. The usual silk sutures were used to close the incision, and the dry carbolic gauze dressing with adhesive straps was applied. No drainage. The operation lasted nearly an hour and the patient was slow in completely rallying, the pupils remaining dilated for nearly an hour after she was placed in bed. The vaginal temperature just after the operation was 97·4° and the pulse 88.

The tumour weighed 1 lb. 11 oz., the greater part being a dark red serum with much cholesterine floating in it. The upper part (about a third in bulk) was the unaltered spleen, the lower part a large globular cyst with the little hernial sac already mentioned projecting from its surface. At its upper part were several pouches of irregular shape and size, projecting into the splenic tissue.

The patient was treated just as if ovariotomy had been performed, *i. e.* she had 3 oz. rectal injections of strong beef-tea every three hours, with twenty drops of laudanum in every other injection. Sickness was troublesome for twenty-four hours, and then she began to take a little

clear beef-tea and bread by the mouth. There was a trace of albumen in the first urine and then it became loaded with urates, and remained so till convalescence was fairly established on the fifth day after operation. On the operation evening the temp. rose to 101·6°, pulse 120, resp. 32. On the next day the highest point was a degree lower, with corresponding fall in pulse and resp., on the second day another degree lower, on the third day it was stationary, and on the fourth day it was normal, with a pulse of 92. A sharp metrostaxis came on on the evening of the second day and ceased on the evening of the fourth day. The only unusual symptom was pain about the pedicle accompanied by occasional difficulty in breathing for the first few days.

The bowels were cleared on the sixth day by enema, and the sutures were removed on the seventh day, the wound having united well by first intention. On the eighth day she was carried down into the convalescent ward, and the move was followed by a slight rise of temp. 100·8°, pulse 104. On the ninth day she was a little sick, and then continued to make an ordinary recovery, with practically normal temp. and pulse, till she got up on the eighteenth day after operation. Two days later the temp. rose and pulse quickened, and there was much pain over the pedicle; this continued more or less, and she was occasionally sick for about ten days, then she had occasional chills and a nose bleeding, and no progress was made. On the thirty-first day the temp. reached 103·4°, with pulse 120; on the thirty-second day it was 104·2° for a few hours, with a pulse of 128, it then suddenly fell, and in two more days was normal. Then in a few days there was a slight relapse, and slight phlebitis in the left leg; this passed off quickly, and she was up again, and after remaining some time in hospital, for fear of a relapse, went to the convalescent home quite well on the sixty-fourth day after operation.

During the convalescence the blood was examined occasionally, and at first there was a slight excess of white

corpuscles, but there was never any perceptible enlargement of the thyroid, or of any of the lymphatic glands. She is now in perfect health, and able to do her work as a domestic servant. I have not seen her, but hear from those who have, that she has a good colour, is stout, and in all respects healthy. The tumour was shown at the Pathological Society, and all that I have to say as to its pathology will be found on page 385 of the thirty-fifth volume of the 'Transactions.'

On July 23rd, 1884, E. M—, married, æt. 25, mother of three children, was admitted under my care at the Samaritan Hospital on the recommendation of Drs. Herman and Turtle, believed to be suffering from an ovarian tumour.

Condition.—Healthy-looking brunette, with bright fresh-coloured cheeks. Tongue furred, appetite bad, much troubled with flatulence, bowels very costive, has been unable to lie down for the last three weeks from pain in both hips; lungs and heart healthy, urine pale and of low specific gravity, but not albuminous. Menstruation at long intervals, and then has profuse and prolonged discharge. The last period lasted for five weeks.

Family history.—Father, mother, and one brother died of lung diseases, and another brother of brain disease; three other brothers and two sisters are healthy.

History.—After birth of last child, a year and a half back, had low fever with diarrhœa, which laid her up for three months. Just after this she first noticed a hard lump in her left side; this enlarged downwards, and is still growing fast.

Examination.—The abdomen is greatly distended with a firm elastic swelling which occupies the whole of the left side of the cavity, and extends below the umbilicus some distance into the right side; this portion of the swelling is covered with intestine. The left flank is dull right back to the spine. The tumour is trilobed; the upper, smaller, and harder lobe lies partly under the ribs on the left side, and the middle and larger lobe extends

from half way between the ensiform cartilage and the umbilicus, down to the left iliac crest and pubes; the third lobe is partly divided from this by a distinct notch at the navel, and extends chiefly to the right of the linea alba. Both these lower lobes are much softer than the upper one, and give an indistinct sense of fluctuation. The lower portion of the tumour is found by vaginal examination to occupy the whole pelvis, pushing the uterus upwards and somewhat behind the pubes. The uterine cavity measures two and a half inches, and there does not appear to be any close connection between this organ and the tumour.

Diagnosis.—Very doubtful; it is more like a cysto-sarcoma of the mesentery that I once removed than anything else, or an inflammatory retroperitoneal tumour. Spleen and kidney cannot, however, be excluded.

On July 23rd, 1884, I made an exploratory incision outside the left rectus (Langenbüch's), as I thought that would give me better access to the deeper parts of the growth. On fully exposing the tumour it was at once evident that it was a case of greatly hypertrophied spleen, and encouraged by the success obtained in the case recorded above, I determined to remove it. The pedicle was very broad, but thin and membranous, containing enormous vessels. The pelvic portion was dislodged with some difficulty, and the omentum was extensively adherent all over its anterior surface. I separated the latter, cutting each separate portion between two ligatures, as the vessels passing between the spleen and omentum were, many of them, large. I then transfixed the pedicle in two places, locking the three ligatures, and tying the outer loop first, then the inner, and the middle one last. Before cutting away the tumour, I put on two large curved pressure forceps so as to secure the main vessels if the ligatures were not tight enough. I then cut the tumour away, put a separate ligature round the whole pedicle, and sponged out the peritoneum. There was no hæmorrhage and everything seemed perfectly secure.

While I was putting in the sutures, some dark blood began to ooze up beside the flat sponge, and when I moved it the whole omentum and mesentery seemed suddenly to have filled with blood, the pressure being so great that the vessels burst as we watched them, and the blood was effused into the cellular tissue. At the same time, the patient's face became deeply congested, and then the parietal peritoneum and the edges of the incision became purple and oozed all over. I pulled up the pedicle which had been dropped and could find no bleeding point, but applied another ligature a little behind the others and round the whole. Finding it impossible to check the general oozing, I rapidly finished the operation, hoping that the condition would pass off, and the circulation become natural, and that the effused blood might then be reabsorbed. The pulse was very bad and flickering at this time, but steadied soon after she was placed in bed to 104, and shortly after was quite good at 96. Her appearance also became normal. She was in bed at 4.15, and at 5.30 a cold perspiration broke out, and pulse and temperature rose quickly. Two ounces of urine were obtained from the bladder at 7. At 9 the temperature was 102·2°, and the pulse hardly to be counted. At 9.45 she died quietly.

Mr. Malcolm made a post-mortem the next day, and found that a very small artery had retracted from the middle loop of the first ligatures, and great hæmorrhage had taken place between the layers of the omentum, and so completely behind the pedicle and exposed parts that it could hardly be seen till they were removed. I conclude that the suffusion of face and general congestion were due to pressure of this enclosed blood upon the sympathetic plexuses causing paralysis of the vessels, the condition passing off when the sac burst and the blood became more generally diffused. In this connection it is interesting to note the condition of my first case while the pedicle was dragged upon by the tumour and also the attacks of

dyspnœa with pain about the pedicle during the first few days after operation.

The mistake I made was in tying the two outer loops of a locked chain before the middle one, as when I tied the latter there were two fixed points on each side of it, and the small membranous portion of the pedicle which it enclosed was not sufficiently tightly constricted. My reason for tying the outer and inner loops first was that all the largest vessels were enclosed in these two loops. In face of this sad accident it is useless to speculate on what might have been, but from the ease and rapidity with which the operation was performed, the perfect immunity from hæmorrhage in separating the adhesions and removing the tumour, and the satisfactory condition of the patient till the hæmorrhage occurred, I think there is every probability that the operation would have been successful. I should not hesitate to operate if I met with a similiar case with symptoms equally demanding relief.

There are now a sufficient number of successful splenectomies on record to show that in proper cases it is a justifiable operation, and if it stood alone my first case would prove that not only is recovery possible, but that the removal of this organ when diseased is followed by a marked improvement in health and by no troubles which can be associated with the loss of the organ.

The following tables give all the cases of splenectomy which I have been able to find, and I have to acknowledge with thanks much assistance from my friend Dr. Pinter, of Pesth, in collecting them. Credé gives them nearly all in a table in a paper published in 'Langenbeck's Archiv,' vol. xxviii, p. 404, but makes a curious mistake in attributing a case to Baker Brown in 1881, *i. e.* eight years after he died. He omits the case by the same operator in 1866, so possibly it is only a mistake in the date. Credé gives leukæmia as the disease for which the operation was performed; my authority, the late Dr. Tanner, says that it was hypertrophy.

It is quite clear from an analysis of these tables that

cases in which the spleen is either itself injured or merely protrudes through a wound in the side, generally do well if treated by complete removal of the organ, or by removal of the injured or protruding portion.

The removal of "simple wandering" spleens is also a safe operation. One in which hypertrophy was also present was unsuccessful.

All the three cases of extirpation of cystic spleens also recovered.

Simple hypertrophy is a much more dangerous condition, most of the deaths being due to hæmorrhage. The large size of the mass to be removed, and the broad pedicle, with its enormous vessels, expose the operator to such accidents as I have recorded above, but we only want experience and greater care in ligaturing the pedicle to make these cases successful. From the account given by Sir Spencer Wells of his second case I should doubt if Credé was right in giving leukæmia as the disease, and certainly the case in 1876, when I assisted at the operation, and made the post-mortem afterwards, was one of simple hypertrophy. Of fourteen cases operated upon for simple hypertrophy, including the "wandering spleen" named above, ten died and four recovered.

All the cases of leukæmia (thirteen out of the total of thirty-four) died, and they make up the great mortality of the operation, so that it is quite clear that when this disease is present it is not justifiable to operate. Excluding them the mortality is still nearly 50 per cent., but it will doubtless be much lower with care in dealing with the pedicle, and with increased experience.

To these complete splenectomies we may add four cases in which an injured spleen was partly removed; all recovered.

Twelve of the thirty-four splenectomies have been performed in Great Britain, and my first case is the only successful one. Italy is to the front with four cases with only one death.

Successful Splenectomies.

No.	Date.	Operator.	Place.	Disease.	Reference.
1	1549	Zacarelli	Naples	Hypertrophy	'Tanner's Practice of Medicine,' vol. ii, 6th ed., 1869, p. 151.
2	1711	Feirerius	St. Carignan	Spleen lying in a peritoneal abscess	'Opuscula Medica et Physiologica Fantoni,' Geneva, 1738.
3	1855	Schultz	Darmstadt	Spleen protruding from wound in side	
4	1867	Péan	Paris	Cyst	'L'Union Médicale,' p. 840, Paris, Nov. 26, 1867.
5	1876	Péan	Paris	Hypertrophy	'Clinique Chirurgicale,' 1875-6, Paris, 1879.
6	1877	Martin	Berlin	"Wandering"	'Brit. Med. Journal,' 1878, vol. i, p. 191.
7	1878	Czerny	Heidelberg	"Wandering"	'Wiener med. Wochensch.,' vol. xxix, 1879.
8	1878	Volney d'Orsay	America	Hypertrophy	'Albert's Lehrbuch der Chir.,' vol. iii, p. 472.
9	1881	Franzolini	Udine	Hypertrophy	
10	1881	Credé	Dresden	Cyst	'Langenbeck's Archiv,' vol. xxviii, 1883, p. 404.
11	1884	Knowsley Thornton	London	Cyst	'Trans. Pathological Soc.,' vol. xxxv, pp. 385-6.

Successful cases of Partial Removal of Injured Spleen.

1	1678	Mathias	—	Was well 6½ years after	
2	1738	John Ferguson	—	Complete recovery	'Philosophical Trans.,' vol. ix, p. 149, London, 1747.
3	1815	Leuhossek	—	Was well 3 years after	Hecker's 'Annalen,' Berlin, 1828.
4	1844	Berthet	—	Lived 13 years	'Archives Générales de Médecine,' 1844, p. 510.

Unsuccessful Splenectomies.

1	1826	Quittenbaŭm	Rostock	Hypertrophy	'Commentatio de Splenis Hypertrophia, &c.,' Rostock, 1826.
2	1855	Küchler	Darmstadt	Hypertrophy	'Extirpation eines Milztumars,' Darmstadt, 1855.
3	1865	Spencer Wells	London	Hypertrophy	'Abdominal Tumours,' 1885, pp. 182—189.

No.	Date.	Operator.	Place.	Disease.	Reference.
4	1866	Bryant	London	Leukæmia	'Guy's Hospital Reports,' 3rd series, vol. xii, p. 444, London, 1866.
5	1866	Baker Brown	London	Hypertrophy	Tanner's 'Practice of Medicine,' vol. ii, p. 151, 6th edition, 1869.
6	1867	Bryant	London	Leukæmia	'Guy's Hospital Reports,' 3rd series, vol. xiii, p. 411, London, 1868.
7	1867	Koeberlé	Strasburg	Leukæmia	'Gazette Hebdomadaire de Médecine et de Chirurgie,' p. 680, Paris, Oct. 25, 1867.
8	1873	Urbinato	Cesana	Hypertrophy of wandering spleen	
9	1878	Koeberlé	Strasburg	Hypertrophy	
10	1878	Spencer Wells	Birmingham	Hypertrophy	See above, Case 3.
11	1878	Heron Watson	Edinburgh	Leukæmia	
12	1876	Spencer Wells	London	Hypertrophy	See above, Case 3.
13	1877	Billroth	Vienna	Leukæmia	'Wiener med. Woch.,' 1877, No. 5.
14	1877	Billroth	Vienna	Leukæmia	
15	1877	Langley Browne	—	Leukæmia	
16	1877	Fuchs	Béhas	Leukæmia	Credé's table, Case 20.
17	1877	Simmons	Sacramento	Leukæmia	
18	1878	Czerny	Heidelberg	Leukæmia	'Wiener med. Wochenschrift,' vol. xxix, 1879.
19	1878	Arnison	—	Leukæmia	'British Medical Journal,' 1878, vol. ii, p. 723.
20	1878	Geissel	Essen	Leukæmia	
21	1881	Haward	London	Leukæmia	'Clinical Society's Trans.,' 1882, and 'B. M. J.,' vol. i, p. 462, 1882.
22	1883	Spanton	—	Hypertrophy	'British Medical Journal,' 1884, vol. i, p. 14.
23	1884	Billroth	Vienna	Sarcoma	
24	1884	K. Thornton	London	Hypertrophy	Now first published

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' New Series, vol. ii p. 103.)

ON THE

DEVELOPMENT OF MAMMARY FUNCTIONS

BY THE

SKIN OF LYING-IN WOMEN.

BY

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The subject of numerical abnormalities of the breasts and nipples has from time to time received considerable attention under the titles of supernumerary mammæ and nipples; it has been referred to by Sir James Simpson,[1] it has been treated by Dr. Mitchell Bruce[2] in an excellent paper, and, most exhaustively, by Professor Leichtenstern.[3] Cases have also been recorded by Dr. Handyside,[4] by Dr. Matthews Duncan,[5] by Mr. Cameron,[6] and by others.

The cases recorded by these observers have included numerical abnormalities of nipples, of nipples with mammary glands, and of mammary glands with pores and without nipples. With these we are now only indirectly

[1] 'Obstetric Works,' vol. ii, p. 325.
[2] 'Journal of Anatomy and Physiology,' vol. xiii, 1878-9, p. 425.
[3] 'Virchow's Archiv,' Band 73, 1878, s. 222.
[4] 'Journal of Anatomy and Physiology,' vol. vi, 1873, p. 56.
[5] 'Obstetrical Journal,' vol. i, 1873, p. 516.
[6] 'Journal of Anatomy and Physiology,' vol. xiii, 1878-9, p. 149.

concerned, indeed, only so far as to include certain specimens which have come under my personal observation, and which serve as a contrast to those cases which I propose to describe, cases which, so far as I know, are new.

These supernumerary structures, described by many authors, are in the great majority of cases situated below the normal mammæ and are a little nearer to the middle line; when they are *above* the mammæ they are always (says Leichtenstern) *more external* than the normal mammæ. But this situation is so rare that out of 105 cases collected by him only 5 were situated in the axilla, while 2 were on the back and 1 on the acromion.

In the case of the five axillary mammæ (S. 254) they were all provided with nipples, often more or less rudimentary, from which milk or colostrum exuded. In 2 cases the side affected was the left; in 3 both sides were affected. Thus, the left side was affected in all in 5 cases, the right in 3.

Mr. Cameron's case, which is not included in Leichtenstern's 5 cases, and, indeed, is somewhat different, is briefly as follows: A married woman, æt. 33, pregnant with her sixth child, observed a swelling under the left arm after over-exerting herself at a fire, when in her alarm she seized several buckets and carried them till she was exhausted. After her confinement milk could be squeezed from the tumour.

When examined a soft tumour was found in the left axilla behind the fold of the pectoralis major; the mass was easily moveable and not connected with the breast of the same side. Its boundaries were difficult to define as the edges appeared to go under some structure and elude the fingers, reminding one somewhat of a hernia. This seemed to lead to the inference that there was originally a capsule or investing membrane which had burst on the occasion mentioned above as a result of over-exertion. This appeared all the more probable as no tumour was suspected before that occurrence, and from its size when examined, and the intelligence of the patient, this seemed

hardly credible, unless some change then took place in its condition or surroundings.

The length of the tumour may be roughly stated at about three inches by about one and a half in breadth. The skin over it was slightly darker in tint than that in the neighbourhood. The tumour was not painful or tender, nor had it given any trouble while suckling the last child. At the time the examination was made the patient was again pregnant, and milk could be drawn from the breast. A small orifice was found at the upper and anterior part of the tumour (but nothing like a nipple); from this a fluid could be squeezed which under the microscope proved to be milk, thus showing the true nature of the tumour.

Since the patient was under observation she has been confined, and it was observed during lactation that milk flowed freely from the tumour, and that whenever the breasts were allowed to become full the tumour swelled coincidently.

Cameron quotes a case related to him by Mr. Bickersteth, in which a somewhat similar tumour, as large as a cricket ball, was removed from the right axilla; it had at first been as large as a walnut and had steadily increased in five years to the size of a cricket ball. It was removed from a distinct capsule and proved to be an adenoma, such as is found only in mammary tissue. The conclusion was, therefore, that the tumour was an adenomatous supernumerary mamma.

The value of this case lies in the microscopical examination, but the great increase of the tumour in size was pathological.

Mr. Bickersteth (says Cameron) had observed a somewhat similar case in an unmarried woman, æt. 33. The tumour was about the size of a fist; it had developed with the development of the breast, but had not increased in size since puberty. It was not interfered with.

Leichtenstern found the left side much oftener affected with supernumerary mammæ and nipples than the right

side in the proportion of seven to two. He remarks that he cannot explain this, but that the left breast is usually the larger. Dr. Mitchell Bruce found them also more frequently on the left side.

The cases which I have to describe were observed in the General Lying-in Hospital and (not including cases like some of those above) number thirty, three of them during Dr. Williams's months of office. He has kindly allowed me to incorporate them.

The first included in the series concerned a patient admitted Oct. 9th, 1882, and the last concerned a patient admitted Nov. 27th, 1884. During this time the total number of patients observed in the hospital was 712. As will be seen by the table annexed, the appearances were observed far more frequently during some periods than others, they were not uniformly distributed in time, and no percentage represents the facts accurately. Indeed, the regular observations began Oct. 1st, 1883; this makes the total number of patients during this series 377, in 27 of whom these swellings were found.

As to the side affected—the right side was affected in 14, the left in 1, both in 15. Thus the total number of times in which the right side was affected was 29, the left 16. This is at variance with the proportion observed in supernumerary mammæ and nipples.

When bilaterally situated the lumps in the right side were the larger in 7; the left in 3; they were of equal size in 5. Thus the right side predominated both in frequency and in size.

It now remains to give—

I. A description of these bodies:

1. They are situated in the *skin* of the axilla, which cannot be pinched up freely over them. On attempting to raise the skin, it seems to be tied to the lumps by fibrous septa.

2. They can be raised and isolated from the deeper structures, and are not in the situation or of the shape and feeling of glands.

3. The skin over them is usually quite natural in appearance.

4. They are limited to the hair-covered surface.

5. They are usually soft, and somewhat elastic except when swollen.

6. They are usually somewhat flattened, their vertical diameter being the smallest.

7. They do not possess any nipple, pore, or duct.

8. Their size varies from the smallest perceptible, to that of an egg, or perhaps larger. (As to the comparative size of those in the right and left axilla, see above.)

II. As regards the *course :*

1. They are most commonly first noticed on the third or fourth day after delivery, at the time when the breasts fill. But they can very often be found, if looked for, at the time of labour, and the patient is sometimes conscious of their presence continuously from her first pregnancy.

2. They sometimes, when once established, become larger and occasionally painful at the beginning of pregnancy, sometimes at quickening, sometimes later in pregnancy, but most commonly not until after delivery.

3. Their course during lying-in usually coincides generally with that of the breasts, enlarging and becoming tense and sometimes tender about the third day, softening as the breasts soften, and becoming much smaller, or even almost imperceptible, by the end of a fortnight. As a rule, however, their size and tenseness does not coincide with the diurnal variations of the breasts in this respect.

III. As regards their *secretion :*

1. In the first 11 the mode of obtaining the secretion had not been discovered.

2. In the remaining 19 (with one exception) secretion of some kind was obtained.

3. In no case did secretion flow spontaneously, as described in some cases of axillary mammæ.

4. To obtain secretion it was necessary to firmly squeeze the lump between the fingers, from the deeper and towards the superficial aspect, as in evacuating a comedo.

5. The secretion was of three principal kinds:—(*a*) Granular débris, like the secretion of sebaceous follicles; (*b*) colostrum; (*c*) milk.

6. The above was usually the order in which the various secretions appeared.

7. Colostrum, milk, and granular débris might disappear and reappear within a few days.

8. At the same time various follicles would produce various secretions. The whole lump was not always uniform in its secretions at the same time.

9. The secretion was expressed from the situation of the sebaceous follicles as marked by the situation of the hairs. Before the secretion exuded for the first time from a follicle which was being squeezed, the follicle was usually seen to swell up, become prominent, whitish in colour, and often to discharge a fluid like thin gum, after which other secretions might follow.

10. The whole surface of the lump produced secretion; there was no centralisation.

In one case (No. 200, admitted August 25th, 1884) belladonna seemed to soften the lump and to promote escape of secretion, as in the case of the breast.

In order to reduce scepticism to a minimum, invitations were sent to many competent observers, and the appearances were seen by Drs. Braxton Hicks, Matthews Duncan, Gervis, John Williams, Herman, and Mr. Clutton, as well as by the author and by Drs. E. S. Tait and Boxall, who were successively house physicians, and from whose careful notes I quote below.

The following well-marked cases are described at length; the main facts of the others are set forth in the table.

Axillary Lumps without Nipples or Pores.

No. 200.—Admitted August 25th, 1884, æt. 30, 3-para. Lumps in both axillæ were noticed on admission.

On the second day the following note was taken:

"In the right axilla at the apex, extending in about

equal proportion on the inner and outer wall, is a lump in the skin three inches long, one and a half inches wide, and three quarters of an inch thick, thicker towards the chest than elsewhere; of even contour; firmly united to skin, and freely moveable on subjacent structures. Skin cannot be pinched up over it, but can be brought together under it, except where it is too thick to allow of it. Surface is covered by a few hairs; hair-covered surface is co-extensive with lump. Not painful, but a little tender on manipulation. No redness of surface, no duct to be seen. On squeezing, a little fluid exudes from a follicle with a hair in the centre, and others swell up, but do not rupture. In the opposite axilla is a similar lump, to which the above description equally applies, except that it is less defined and somewhat softer. Patient first noticed the lumps two or three months before her first confinement; smarting in the armpits drew her attention to them. They were smaller then than now; they got bigger and more painful till confinement and then went away, beginning to get smaller directly after labour, and had entirely gone at the end of a month. They were never then as large as now. The same series of events happened in the second pregnancy and after labour, but the lumps were larger than before and more painful. In this pregnancy they were noticed first about eight months ago, the aching pain drew attention to them, and she thought an abscess was forming. They have gradually got bigger and more tender up to the present time."

On the fourth day the lumps were noticed to be rather harder and more tender. Glycerine of belladonna applied.

On the fifth day the lumps were relieved by the belladonna. "On squeezing the lumps the follicles of the skin over them enlarge, and fluid oozes up around the hairs."

On the seventh day, "the lump under the right arm was squeezed, the follicles swelled up and fluid exuded around the hairs. This was collected from three different follicles; from one it came in great abundance and looked quite like milk both to the naked eye and under the

microscope; it was perfectly typical, with a few colostrum corpuscles. Another specimen from another follicle showed many very well-formed colostrum corpuscles and milk globules, and a third specimen from another follicle showed a few colostrum corpuscles and a few globules like dilute milk. Of its character there can be no doubt."

On the ninth day the lumps were softer, smaller, and much less tender. "Milk from the other (left) lump was examined microscopically. It proved to be typical milk with excellent colostrum corpuscles."

On the tenth day, "the lumps keep much the same. When asked if they are still painful, patient volunteers the information, 'Only when the draught comes into the breast, they get hard at the same time, but subside with the breast.'"

On the fourteenth day, "says the lumps get hard at night when the child is put to the breast, but soon subside when the breast is emptied. Knows when she is in the family way by pain being felt in the lumps."

No. 239.—Admitted October 16th, 1884, æt. 33, 4-para.

On the second day the following note was made:

"In either axilla is a soft lump in the skin, so soft at present that it cannot well be defined. That on the left side is harder than that on the right; they are limited to the hair-covered surface. The skin beneath them can be nearly, but not quite, pinched together, as the lump is of considerable thickness; it cannot be pinched up upon it. No redness, throbbing, pain or tenderness. No duct can be seen. Not noticed before."

On the third day, "each lump is about the size of a large walnut, harder and more defined than yesterday."

On the fourth day, "the lumps rather larger, but not much harder. On squeezing them the follicles in the skin swell and exude fluid."

On the fifth day, "the breasts became hard in the night and so did the lumps. The breasts are now full, and the lumps are hard and well defined.

On the sixth day, "both lumps and breasts softer."

On the eighth day, "the breasts are soft and so are the lumps. The lump in the left axilla was squeezed firmly, and the hair-follicles swelled up as white points, looking something like small pustules with a hair in the summit of each. Some fluid begins to exude around the hairs in seven or eight places. This fluid collected on a cover-glass and examined under a microscope is seen to consist mainly of granular epithelial débris, much of which is freely floating with a few free oil-globules and a considerable number of colostrum corpuscles."

On the eleventh day, "lump in either axilla hardens whenever breast of same side gets hard and full."

On the twelfth day, "microscopic specimen made of fluid from lump in right axilla proves to be similar to that from the opposite side on eighth day. Both lumps are much softer to-day, and the breasts are soft too. Now says she noticed the lumps soon after first confinement, and they ran a similar course. The doctor in attendance also noticed them, and told her she had a small tumour in either armpit, and requested her to go to him again after she got about, but she did not do so."

No. 287.—Admitted November 29, 1884, under Dr. Williams, æt. 39, 8-para.

On admission very soft lumps were noticed in both axillæ, not easily defined at present, that in the right the larger.

On the second day, "at the apex of either axilla, is a lump in the skin, very soft at present, so that its area cannot well be defined. It is commensurate apparently with the hair-covered surface, and is three inches long by two inches broad, the long diameter running from the chest in the direction of the axis of the limb. It appears to be about half-an-inch thick, and is of barely firmer consistence than an accumulation of fat would be. There is no abnormal appearance on the skin, no redness, and no duct. The lump forms a visible fulness in the apex of the axilla. It is of fairly even contour, a little tender on manipulation; the skin can be pinched almost but not quite together under it owing to its extent and thick-

ness, but cannot be pinched up over it; it is freely moveable on the subjacent structures. On squeezing the lump the follicles in the skin over it swell, and a small quantity of fluid exudes from several hair-follicles around the hairs. This effect is produced by very little squeezing. On collecting this fluid from several follicles for microscopical examination, it looks opalescent between glass, and on further examination it is seen to consist mainly of granular débris with a few oil globules of varying size floating freely in a clear liquid, and in another part of the specimen are many globules with several large, well-defined colostrum corpuscles. The lump on the left side is rather smaller and softer than on the right. These lumps were noticed on admission and have become rather larger, harder, and more defined since. She herself was unaware of their existence, and knew of none in her previous pregnancies or lyings-in, but the axillæ have, after each confinement, but not before, "become tender till the flow of milk came in, and I thought it was from throwing my arms about when I was confined."

On the third day, "lumps scarcely altered, perhaps that on right side a trifle harder, that on left side is rather larger and harder, so as to more nearly equal that on the opposite side. Fluid from left expressed and examined in same way as that from right, shows same characters in a much more marked degree. In one portion of the specimen is almost pure granular débris, with here and there a colostrum corpuscle; in another is an innumerable colony of perfect colostrum corpuscles without any admixture; and in another oil-globules of varying size with a few colostrum corpuscles and granular débris intermixed. Dr. Herman saw the lumps this evening, and fluid was expressed from the right and examined by him."

On the fourth day, "lumps the same; it is difficult to separate the breast-gland from the lump on the left side, and on the right they become almost contiguous. The situation between the lumps and breasts on either side is tender."

On the fifth day, "Dr. Matthews Duncan saw the lumps last evening and also saw the same microscopical specimen as Dr. Herman. He thought the colostrum corpuscles were small, and had too defined an outline. A fresh specimen was made in his presence from the lump in the right axilla, and it proved to be milk with two or three of the same kind of corpuscles. Of the milk he had no doubt. The lumps are both rather larger in area and thicker than they were; they measure $3\frac{1}{2} \times 2\frac{3}{4}$ in. (right side); $3 \times 2\frac{1}{2}$ (left side). Their consistence remains unaltered. They are decidedly less tender than they were."

On the sixth day, "lumps same in size and feeling but not tender on manipulation. Dr. Gervis saw the lumps this afternoon. The fluid expressed from the outer portion of the left lump showed under the microscope mainly granular débris with a few oil globules and colostrum corpuscles."

On the tenth day, "lumps are getting decidedly softer. Dr. Braxton Hicks saw the lumps this afternoon. Very little fluid could be expressed, but sufficient for microscopical examination. It proved to consist mainly of granular débris and colostrum corpuscles."

On fourteenth day the patient was discharged, with "the lumps scarcely altered."

The following cases, which were observed concurrently with the others, are here inserted by way of contrast, and to show that the author was on the look-out for all varieties of mammary abnormalities.

A. *Extension of Mammæ into Axillæ.*

No. 16.—Admitted January 17th, 1884.

On third day a projection from the mammæ was observed to extend into the apex of each axilla; its greatest breadth was two inches. It was nodular and in all respects like the breast tissue. It joined the outer border of each breast at a tangent. It was fairly move-

able on subjacent structures, the skin over it was freely moveable and could be pinched up. There was no accessory nipple or unusual appearance in the axilla. No secretion could be expressed.

On the fifth day it was noted that the left breast had been sucked and was soft, and so was the axillary extension; that the right breast was harder, and so was the axillary extension.

No. 146.—Admitted July 6th, 1884.

On sixth day the following note was taken:

"In either axilla on the inner wall is an extension of the mamma as far as the apex, it is soft and feels like mammary substance, evidently connected with the breast, and freely moveable on the deep structures. The skin can be pinched up over it. No duct or nipple can be found." No secretion could be expressed.

No. 156.—Admitted July 11th, 1884 (see also "axillary lumps").

On second day it was noted that in each axilla, running up from the side of the breast along the inner wall towards the apex, was a prolongation of the breast, glandular and nodular in feeling, and softer than the "axillary lump" in the skin of the right axilla, which it met at an angle at the apex of the axilla. No duct or nipple could be found and no secretion expressed.

B. *Separate Axillary Mammæ with Axillary Nipples, Pores, or Ducts.*

No. 136.—Admitted June 27th, 1884.

On the second day the following note was taken:

"In either axilla at the apex is a supernumerary mamma. That in the right is more distinct and as large as a pigeon's egg, at present soft and tender. A tail from this runs down the arm half an inch to an inch, and is a little harder than the rest. The skin can everywhere be pinched up over it, and it is fairly moveable on the subjacent structures. It opens by a duct in the anterior

axillary fold, the opening projects slightly, is perhaps faintly erectile, and out of it a bead of juice can be expressed. A similar lump is found in the opposite axilla with the following differences:—It is softer; has no tail, and out of the duct colostrum can be pressed. No colostrum can be obtained from either breast."

On the third day colostrum could be squeezed from both axillary mammæ.

On the fourth day both were rather harder and distinctly nodulated like breast substance; milk could be squeezed from both.

On the sixth day the right axillary mamma was larger, and a second pore was found, from which milk could be squeezed.

No 152.—Admitted July 10th, 1884.

On the third day the following note was taken:

"In the right axilla is a lump which feels glandular, rather softer than the breast of the same side, nearly the size of a pigeon's egg, but too soft to define. It runs from the apex towards the deep structures at the margin of the breast, its surface is covered by hair, it is freely moveable on the subjacent structures, the skin can be pinched up over it. At the anterior border, *i.e.* at the anterior axillary fold, a minute duct can be found, especially on pinching up the skin, when it becomes retracted in that spot; it projects slightly, and is of a little more pigmented colour than the surrounding skin. Scarcely any moisture can be expressed from it. It is not painful. In the opposite axilla is a similar body, but softer and half the size, with a less distinct duct in a corresponding situation. She had a painful lump in either axilla three days after her first confinement, it went away when the milk was dried up a week later."

On the seventh day milk was expressed from the duct in the anterior fold of the right axilla.

No. 106.—Admitted May 24th, 1884, under Dr. Williams.

On the third day the following note was taken:

"In the left axilla, on the costal wall, close to the apex,

a lump can be felt just beneath the skin, which can be pinched up over it except at one spot where there is a minute hole, a little pinkish and pigmented, just visible to the naked eye, but its position is readily ascertainable by pinching up the skin over the lump, when a dimple is produced at the spot, showing it to be bound down to the deeper structures in that situation. There is no projection of the surface. The lump is about the size of half a nutmeg, round, freely moveable on the deeper structures, and apparently continuous with the glandular substance of the breasts, the connecting medium being an isthmus about one inch long, one third of an inch broad, and one third of an inch thick. The consistence of the isthmus and of the lump corresponds with that of the breast, undulating on the surface. None was found in the left axilla on examination yesterday, and none is apparent now in the right axilla. On squeezing the lump, out of the small pore a drop of fluid was expressed, which the microscope showed to be milk and colostrum."

On the fifth day, "in the right axilla is a small papilla corresponding in situation to that on the opposite side, and a little more distinct than it, standing up one sixteenth of an inch above the surface, and of a brownish-pink colour. In the centre is a duct, out of which milk can be squeezed. It is attached to something beneath, like that on the opposite side, but no lump can be felt. The margin of the breast is distant about one inch."

C. *Supernumerary nipples (without special gland substance).*

No. 301.—Admitted December 12th, 1884, under Dr. Williams.

On the third day the following note was taken:

"Immediately below the left nipple, one and a half inches from the lower margin of the breast, is a nipple-like wart, as large as a pea, with a small pedicle, quite short, and surrounded by a bronzed areola one sixteenth of an inch

wide. No opening can be found in it, there appears to be no gland tissue beneath."

No. 317.—Admitted November 28th, 1883.

On the fourth day the following note was taken:

"Below each breast is a pigmented wart suggestive of a supernumerary nipple. That on the left side is vertically below the nipple and situated on the costal arch; its diameter is about an eighth of an inch; it projects about a sixteenth of an inch from the surface, has a central depression and is surrounded by a pigmented area. It is distinctly erectile on irritation, but no moisture exudes on pressure. On the right side is a similar body midway between the nipple and costal arch, that is, lying over about the seventh rib, two and a half inches from the costal margin, and one and a half inches from the circumference of the breast. It is like the other in all respects, but is about twice as large, and a serous moisture exudes on pressure. No gland substance can be felt, nor any elevation of the skin. Says her sister has similar bodies. Thinks they are a little darker than they used to be."

Copy of a letter from patient's sister:

"I have only one small, round place about the size of a small pea, smooth and a brown colour, a small hole in the middle and just below the left breast. I believe I have had it from my birth; not like a nipple."

No. 181.—Admitted August 8th, 1884.

On the second day the following note was taken:

"At the lower margin of each breast, almost vertically below the nipples but one inch towards the middle line, is a rudimentary nipple projecting about one sixteenth of an inch, consisting of distinctly erectile tissue, of brownish-pink colour and faintly pigmented around for a quarter of an inch. Each has a depression in the centre and looks exactly like a diminutive nipple. That on the left side is a little the more pronounced. There is no swelling beneath to indicate gland substance. On drawing up the skin, the depression in the centre becomes very evident."

No. 186.—Admitted August 10th, 1884.

This was a remarkable case, having a typical "axillary lump" (see Table) in the skin of the right axilla, and *also three small axillary lumps on the right side, and three rudimentary nipples, two on the right side and one on the left.*

On admission the following note was taken:

"At the circumference of the right breast, vertically above the nipple is a small rudimentary nipple of pinkish-brown colour, apparently erectile, with a dimple in the centre, made most distinct by pinching up the skin; at the side and at the lower border is a still smaller but similar structure. Three axillary lumps close together in the right axilla, none in the left, each the size of a cherry stone, on the outer wall, close to the apex. The skin over them is red (says it feels tender when washed); the follicles on the surface are distended, and become more so when squeezed, and ultimately give way in several places, exuding slightly opalescent fluid, which under the microscope is seen to consist of granular and fatty epithelial detritus."

On the second day an indistinct axillary lump was felt in the right axilla in addition to those described above (see Table).

On the 9th day the following note was taken:

"Lumps in axillæ gone. At the circumference of the left breast also (see condition on admission) is a still more marked rudimentary nipple, situated vertically below the nipple, of a brownish-red colour, decidedly erectile, with a depression in the centre, out of which milk readily exudes (confirmed by microscope). None obtained from the others described on the right breast."

No 196.—Admitted August 23rd, 1884.

On the second day the following note was taken:

"Three inches and three quarters vertically below the right nipple is a rudimentary nipple of brownish colour with a faint areola round, and slightly erectile, with a depression in the centre. None on the opposite side. No secretion."

No. 198.—Admitted August 25th, 1885.

On admission a rudimentary nipple was found at the upper margin of the right breast.

No. 200.—Admitted August 25th, 1885.

On admission a doubtful rudimentary nipple was found at the lower margin of the right breast.

The cases which I have described, and which I believe have not been hitherto recognised, seem to prove that in lying-in women the sebaceous follicles of the skin are capable of producing true mammary secretions. The transition from granular material, through colostrum to true milk, is distinct and unmistakeable. They confirm the opinion that the breast is a highly specialised aggregation of highly specialised sebaceous follicles. The least specialised form (1) is that here described, where the skin is merely thickened, and the sebaceous glands may produce true mammary secretions. The next form is (2) that where there is an aggregation of the ducts, which is open by one or more external pores. The highest rudimentary form (3) is where a nipple, or more, is superadded to the last variety. It is also well known that nipples may be developed independently.

I have not yet had an opportunity of making a microscopical examination, but these structures are so far from rare that, when attention is once directed to them, opportunities are sure to arise sooner or later. The secretions were too scanty for chemical analysis. It is far from improbable that they may share the pathological affections of the breast, and even be the seat of abscess.

Verneuil has described lumps in the skin of various parts of the body, which he concludes to be situated in the sweat-glands. One of the favourite places is the axilla, another the mammary areola. Other situations, such as the region of the anus, are not like those which I have described. Not a word is said of any secretion, nor of their connection with pregnancy and lying in. Some parts of their characteristics are never-

theless so much like those above that they are given below. It will be seen, however, that the details are comparatively scanty, and that their situation remained a matter of opinion.

Verneuil's papers are to be found in the 'Arch. gén. de Méd.,' v série, tome 4, 1854, p. 447. ("Études sur les tumueurs de la peau; de quelques maladies des glandes sudoripares.")

(Ibidem, ibidem, p. 693.)

(Ibidem, vi série, tome 4, p. 537.) Sudoriparous abscesses are common in the mammary areola, in the axilla, and round the anus. They were called "abscès tubériformes ou tuberculeux" first by Velpeau, but their seat was unknown to him. Verneuil calls them "Hidrosadénite."

(Ibidem, ibidem, p. 542.) These abscesses are rarely idiopathic, and are nearly always secondary to local or general causes. Predisposing conditions are to be found in the acrid and profuse sweats of the axilla, anus, scrotum, &c., especially in hot weather.

(Ibidem, ibidem, p. 544.) In the absence of local causes the affection may be due to general causes, such as scrofula. It is equally common in the two sexes; it is common in adult life.

(Ibidem, ibidem, p. 545.) The affection may be situated anywhere except on the palms of the hands and soles of the feet, where the thickest part of the epidermis seems antagonistic to it.

(Ibidem, ibidem, p. 546.) In the axilla, where the sudoriparous glands are most developed, their size may equal that of a pigeon's egg.

(Ibidem, ibidem, p. 547.) They are isolated from the deep parts of the axilla by fascia, whereas the skin is distensible.

(Ibidem, ibidem, p. 548.) Septa pass from the skin to the fascia.

(Ibidem, vi série, tome 5, p. 327 and p. 437.)

P. 442. If the skin be loose, thin, and movable, the

induration can be raised in a fold between the finger and thumb . . . . ; if the induration be somewhat extended the skin can be pinched up in front of it; it is painless or only causes very slight prickling. Direct pressure, or pressure between the fingers on the other hand, is painful.

The following case, for which I am indebted to Dr. John Williams, suggests that these axillary lumps may be subject to the same sympathetic affections as the breasts.

E. C—, æt. 22, married, had one child twenty months before she was seen on September 14th, 1885, at University College Hospital, complaining of pain and a discharge apparently the result of inflammation after her confinement.

She spontaneously complains of a little pain in the left axilla. When she was between nineteen and twenty she thinks she had a small swelling there during a menstrual period.

In the last month or two of her pregnancy she had pain in the left axilla and felt a lump there; it went away soon after her confinement. At the present time her attention has been again attracted to the same spot, but she has been unable to find any lump. She is now near the end of a menstrual period, and a little thickening of the skin covered by the hairs can be felt.

During her pregnancy she had pain in the left breast, but not in the right, and during the present menstrual period she has had it again. As a rule she has had no pain in the breast except at the menstrual period.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' New Series, vol. ii, p. 106.)

No. of case	Hospital No.	Date of admission.	One or both sides.	Dimensions (inches).	First noticed.	Largest which day.	State when last seen.	Secretion.	Remarks.
1	253	Oct. 9, 1882	Both	Right bilobed, size of a walnut; left much smaller	About time of quickening	—	16th day nearly disappeared	—	7-para; lumps first noticed about time of quickening; grew till delivery, and became tender; size and tenderness diminished since. The state of repletion of the breasts produced no effect on their size.
2	255	Oct. 11, 1882	Both	Right size of half a pigeon's egg; left half that size	3rd day	—	Much smaller on 7th day	None	2-para; first noticed at 7 months; increased till delivery, decreased since; same in last pregnancy, disappearing three months after delivery. State of repletion of breasts does not affect lumps.
3	289	Nov. 21, 1882	Both	Right 3 × 1; left same	3rd day	3rd day	14th day fast disappearing	None	Small mole in left axilla, from which nothing can be squeezed; 7-para; similar lumps in all former confinements, beginning about 3rd day, lasting a few days, varying according to repletion of breasts; this variation not distinctly observed while in hospital.
4	278	Oct. 18, 1883	Both	Left = chestnut; right = bean	5th day	5th day	10th day nearly gone	—	—

5	293	Nov. 4, 1883	Both	Right $2 \times \frac{1}{2} \times \frac{1}{2}$; left $1\frac{1}{2} \times \frac{3}{4}$	3rd day	5th day	14th day still distinct	—	Both lumps bilobed; upper lobe larger.
6	301	Nov. 8, 1883	Right	Billiard ball	2nd day	3rd to 10th day	16th day slightly smaller	—	—
7	306	Nov. 14, 1883	Right	$1\frac{1}{2} \times 1 \times \frac{1}{2}$	4th day	4th to 5th day	14th day somewhat smaller	—	—
8	311	Dec. 4, 1883	Right	$2 \times \frac{3}{4} \times \frac{1}{2}$	2nd day	3rd day	14th day a little smaller	—	—
9	2	Jan. 1, 1884	Right	Rabbit's kidney	2nd day	2nd to 3rd day	4th day half size	—	—
10	36	Feb. 21, 1884	Right	Bean	1st day	1st day	14th day small nut	—	3-para; same in 2nd confinement.
11	134	June 23, 1884	Both	Small, ill-defined, especially in left	2nd day	3rd and 4th days	13th day right nearly gone; 5th day left gone	—	8-para; same after 4th and 5th confinement.
12	147	July 6, 1884	Right	Coffee bean	10th day	10th day	Gone on 12th day	Milk on 10th day from the root of a hair.	—
13	149	July 8, 1884	Right	Nut	Before delivery	3rd to 5th day	13th day much smaller	8th day epithelial débris on pressure from roots of hairs	—
14	151	July 10, 1884	Both	$2 \times \frac{3}{4}$	2nd day	7th day	14th day considerable size	Epithelial débris with a few doubtful colostrum corpuscles exudes on pressure from roots of hairs	Wart in right axilla; right lump larger than left.
15	156	July 11, 1884	Right	$1\frac{1}{2} \times \frac{3}{4}$	2nd day	3rd day	6th day a little softer	6th day milk with few colostrum corpuscles exudes from roots of hairs on pressure	4-para; the same after last confinement. Extension of mammæ into each axilla separate from lump.
16	158	July 13, 1884	Right	Large almond	1st day	3rd and 4th days	10th day disappeared	3rd day granular débris from roots of hairs on pressure	—

No. of case	Hospital No.	Date of admission.	One or both sides.	Dimensions.	First noticed.	Largest which day.	State when last seen.	Secretion.	Remarks.
17	175	Aug. 5, 1884	Both	Right 1 × ⅔; left cherry-stone	1st day	3rd day	14th day, both disappeared	1st day fluid exudes from roots of hairs, on pressure; from some epithelial débris with few oil-globules, from others oil-globules as large as the largest in milk, a few apparently fused, a few small oil-globules, others just like small milk-globules	2-para.; lump in right axilla noticed one month after confinement, never quite disappeared.
18	179	Aug. 7, 1884	Right	Cherry-stone	5th day	No change	14th day no change	5th day milk, small globules, no colostrum	
19	186	Aug. 10, 1884	Right	Small, ill-defined (3 others size of cherry-stone, 4 in all)	2nd day (3 small ones on admission)	3rd day	Gone on 9th day	Sebaceous matter squeezed from roots of hairs on 3rd day (from 3 small lumps on admission)	3 rudimentary nipples, 2 right, 1 left; 4 axillary lumps right.
20	191	Aug. 13, 1884	Both	Right 2⅝ × 1½; left 3½ × 1⅜	2nd day	5th day	On 14th day much smaller	Opalescent fluid squeezed from roots of hairs, consisting of granular and epithelial débris, with oil-globules of various sizes	6-para.; noticed since 2nd or 3rd day after 3rd confinement, always present since. During 1st lactation left lump as large as an egg, hard, and tender, larger during lactation and during great perspiration
21	200	Aug. 25, 1884	Both	3 × 1½ × ⅔	1st day	4th day	14th day still distinct	Typical milk, with colostrum corpuscles from roots of hairs over both lumps	3-para.; lumps first noticed by smarting 2 or 3 months before 1st confinement, increased till confinement and diminished directly after; the same in 2nd pregnancy, but larger and more painful than before. In this pregnancy noticed

									in second month by aching, have grown bigger till now. Pain in lumps 1st sign of pregnancy. Belladonna removed hardness and promoted secretion.
22	206	Aug. 30, 1884	Right	Soft and ill-defined	3rd day	Always ill-defined	8th day no change	Milk-like fluid squeezed from roots of hairs consists of granular and epithelial débris, with a few oil-globules of varying size	5-para; never noticed till found by physician.
23	230	Sept. 19, 1884	Left	Size of Barcelona nut	On admission	2nd day	13th day still distinct	Fluid squeezed from roots of hairs consists of epithelial débris, with many oil-globules of varying size	—
24	234	Sept. 24, 1884	Both	Right $2\frac{1}{4} \times 1\frac{1}{4}$, left smaller	2nd day	3rd and 4th day	14th day still distinct	On 4th day clear. 7th day granular débris, many oil-globules of varying size, and a few small but typical colostrum corpuscles. 14th day granular débris, typical milk, and a few colostrum corpuscles squeezed from roots of hair. From a separate follicle on 14th day granular débris, with few oil-globules	13-para; lump in right side since last confinement, 2 or 3 days after delivery; got harder and softer as breasts did.
25	239	Oct. 1, 1884	Both	Size of large walnut	2nd day	5th day	12th day still distinct	Fluid pressed from roots of hairs consists of granular débris, a few free oil-globules, and many colostrum corpuscles	4-para; noticed after first confinement by patient and doctor; their course was similar.
26	248	Oct. 9, 1884	Both	Right, size of almond, left 3 or 4 times as large	3rd day	Not much change	13th day still distinct	None, but follicles swell up on squeezing	3-para; first noticed a few days after first confinement, became painful and then subsided, the same in the next confinement.

No. of case	Hospital No.	Date of admission.	One or both sides.	Dimensions.	First noticed.	Largest which day.	State when last seen.	Secretion.	Remarks.
27	252	Oct. 13, 1884	Right	Size of cherry-stone	2nd day	4th day	8th day still distinct	A little fluid squeezed from roots of hairs	—
28	287	Nov. 15, 1884	Both	Right $3\frac{1}{2} \times 2\frac{1}{2} \times \frac{1}{2}$, left $3 \times 2\frac{1}{2} \times \frac{1}{2}$	On admission	5th to 10th day	14th day plainly palpable	Fluid squeezed from roots of hairs on 2nd day, granular débris, few oil-globules of varying size and large distinct colostrum corpuscles. 3rd day many colostrum corpuscles. 5th day milk and colostrum. 10th day chiefly granular débris and colostrum corpuscles	8-para; never noticed before, but after each confinement tenderness in axillæ. Lumps and secretion seen by Drs. Braxton Hicks, Matthews Duncan, Gervis and Herman.
29	296	Nov. 27, 1884	Right	Half a nutmeg	2nd day	4th to 6th day	14th day half original size	7th day pure milk from roots of hairs, a few large colostrum corpuscles	7-para; noticed since first confinement; gets larger before confinement, large till about third day after confinement, and smaller when child sucks properly. Between pregnancies size of cherry-stone, but never disappears. Seen by Mr. Clutton.
30	297	Nov. 27, 1884	Both	Right $1\frac{1}{4} \times 1 \times \frac{1}{2}$; left $1\frac{1}{2} \times \frac{3}{4}$	1st day	Left 3rd day; right 4th day	14th day half original size	Fluid squeezed from roots of hairs; third day granular and epithelial débris, few oil-globules and colostrum corpuscles; same on 14th day	9-para; never noticed by patient, but tenderness in axilla always when breasts got full soon after labour and at other times. Had to wean 7th child, and had a painful lump under left arm. Seen by Mr. Clutton.

THE

LIGATION OF THE LARGER ARTERIES IN THEIR CONTINUITY.

AN EXPERIMENTAL INQUIRY.

BY

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I.—*Object of Paper.*

The object of this communication is to show that, in the ligature of a large artery in its continuity, it is neither necessary nor advisable to tie the ligature so tightly as to rupture the coats of the vessel; and, further, to demonstrate that a small round ligature possessed of certain qualities and used with the least possible disturbance of the sheath of the vessel is the best for the purpose.

With reference to the occlusion of the smaller arteries, such as the radial, and to the ligature of the cut ends of arteries large or small in an amputation stump, we are not now concerned. In the former case such vessels do not require any special precautions or methods in order to ensure their safe obliteration, and in the latter the question must be looked at from a different point of view.

II.—*Historical Sketch.*

Centuries before the discovery of the circulation[1] of the blood the ligation of arteries for wounds[2] and aneurisms[3] was practised. A great diversity of opinion has always existed as to the best method of performing the operation. The practice of surgeons from the earliest times to the present day seems to have been based on one or other of two great opposing principles:

[1] Harvey, 'De motu cordis et sanguinis,' 1618.

[2] Celsus (book v, chapter 26, paragraph 21):—"But if pressure and astringents are ineffectual to restrain the hæmorrhage, the bleeding vessel is to be taken up, and a ligature having been applied on each side of the wound in it, the vessel is then to be divided; the two parts of the vessel will become united by anastomosing branches, and the orifices will become obliterated."

[3] Galen (Kuhn's edition, chap. 23, vol. xi, p. 313):—"If the artery be large, and if it be cicatrized beyond the aneurism, the whole of it should be cut through, and oftentimes that very practice prevents the danger from hæmorrhage; for it appears plainly that when a complete transverse division is made both portions of the artery retract on either side, the one above the part, the other below." Paré (Works, 1579, translation by Johnson, 1665, p. 323), was the great advocate of the ligature after Galen. He says, concerning the stanching of bleeding in amputation: "The ends of the vessels lying hid in the flesh, must be taken hold of and drawn with this instrument (forceps) forth of the muscles, whereinto they presently after the amputation withdrew themselves. In performance of this work, you need take no great care, if you together with the vessels comprehend some portion of the neighbouring parts, as of the flesh, for hereof will ensue no harm; but the vessells will so be consolidated with more ease, than if they being bloodlesse parts should grow together by themselves." P. 325:—"Wherefore I must earnestly entreat all Chirurgeons, that leaving this old and too cruel way of healing [actual cautery], they would embrace this new, which I think was taught me by the special favour of the sacred Deity; for I learnt it not of my masters, nor of any other; neither have I at any time found it used by any; only I have read it in Galen, that there was no speedier remedy for stanching of blood, than to bind the vessels (through which it flowed) towards their roots, to wit, the liver and heart. This precept of Galen, of binding and sowing the veins and arteries in the new wounds, when as I thought it might be drawn to these which are made by the amputation of members, I attempted it in many." Ambrose Paré, 1582 (Paré, Works, Lyon, 1641, quoted by Erichsen):—"Divide the skin above the aneurism, and, separating the artery, pass a seton needle armed with a strong thread under it, and allow the ligature to fall of itself. Nature will then generate flesh which will block up the artery."

1. That of tying with considerable force in the belief that damage to the arterial wall was either essential to obliteration or a necessary safeguard against hæmorrhage.

2. That of treating the artery with gentleness in the endeavour to cause its obliteration without inflicting the least injury to it.

The earlier surgical writers, Galen,[1] Paulus Ægineta,[2] and others recommend the application of two ligatures and the division of the artery between them; an operation which now bears the name of Abernethy[3] (1827), but many others have practised it. This way of tying an artery probably originated in the observation that arteries in amputation stumps are less prone to secondary hæmorrhage than those tied in continuity; a fact which explains the favour with which the operation has lately been received, and gives the reason for its attempted revival.[4] The validity of this analogy was questioned by Sir Charles Bell[5] sixty years ago, and the procedure appears unnecessarily severe.

The earlier surgeons belong to the severer school, and with them must be placed Jones[6] (1805), who from experiments upon the lower animals considered that he had demonstrated conclusively that the tunics should be ruptured in tying an artery in its continuity. He advocated also the isolation of the vessel and the use of the small round ligature. He says you must divide the two inner coats because if you do not adhesion will not take place, and, as the ligature ulcerates through, hæmor-

[1] Loc. cit.

[2] Paulus Ægineta (seventh century):—"The artery having been cleared of the surrounding parts is to be exposed with the same scalpels with which the membranes have been divided; a needle being then passed under it, the artery is to be tied with a double ligature, having previously been punctured in the middle; suppuration must then be promoted till the ligatures fall out." ('Observations on Aneurism,' collected and translated by John Erichsen, Sydenham Society, 1844.)

[3] Abernethy, Surgical Works, new edit., 1827.

[4] Walsham, 'Brit. Med. Journ.,' 1883, vol. i, p. 660.

[5] Bell, 'The Great Operations of Surgery,' 1821.

[6] Jones, 'On Hæmorrhage,' 1805, p. 170.

rhage will occur. And again, "I cannot be expected to illustrate these opinions by cases, nor would it be easy to confirm them on dogs, for whom nature does so much." Thus Jones made no experiment upon the effect of *not* dividing the coats; he inferred it from the process of repair in nature in wounded arteries, but *if the coats are not cut the artery is not wounded.* It is upon this insecure basis that the established rule of the present day, with regard to the treatment of the wall of the vessel, rests. The majority of English surgeons adopted the views of Jones. In 1813 and 1815 Travers[1] reported his experiments to this Society and recommended the employment of the temporary ligature, and also, as an indispensable condition of obliteration, the rupture of the tunics.

The milder treatment of the wall of the artery has, however, long had its advocates. Alexander Monro[2] (1725) employed a wide ligature not drawn very tight to avoid injuring the vessel. Benjamin Bell[3] (1787) writes, in his 'System of Surgery,' "There is no occasion whatever for making the ligature so tight on arteries as to run any risk of dividing them; a much less degree of pressure than is commonly applied, or could have any influence in hurting them, being fully sufficient for compressing them in the most effectual manner." The best known advocate of gentleness is Scarpa[4] (1817), who was investigating the subject in Italy about the same time that Jones was at work in England. To him is undoubtedly due the honour of demonstrating that the rupture of the coats of an artery is not necessary for its obliteration by ligature. He employed a tape ligature to avoid damage to the arterial wall, and inserted a cylinder of lint between the ligature and the vessel, so as to flatten the latter. The ligature and cylinder were removed on the third, fourth, or fifth

[1] 'Med.-Chir. Trans.,' vol. iv, 1813, and vol. vi, 1815.

[2] Monro, collected works, 1725.

[3] Bell, 'System of Surgery,' 1787, vol. i, p. 61.

[4] Scarpa, 'Mem. sulla Legatura delle principali Arteri degli Arti, con append. sull' Aneurisma,' 1817.

day. By this method Scarpa and his followers obtained numerous successful results. In this country, however, Jones's views were already accepted, and consequently the Italian surgeon had scarcely any English adherents. But in 1821 Sir Charles Bell[1] published his work entitled 'The Great Operations of Surgery,' and in it directs that "the loop and knot of the ligature be sunk into the coats sufficiently to prevent the pulsation of the vessel shifting the ligature, but not drawn so tight as to cut the inner coats of the artery."

Many years before the discussion between the adherents of Jones and Scarpa (as to the best treatment of the wall of the artery) had become acute, the great advance of cutting ligatures short was attempted. This, it would appear, was first carried out by two assistant surgeons of the Royal Navy, Mr. Lancelot Haire[2] and another at the Haslar Hospital about the year 1780. To Lawrence[3] (1814) is due the development of this practice, which was not wholly satisfactory, for, as in Haire's cases, though the wounds healed by first intention yet subsequently the ligature almost always suppurated out. The next step was the trial by Astley Cooper[4] (1817) of catgut with the ends

[1] Bell, 'The Great Operations of Surgery,' 1821.

[2] Lancelot Haire, 'London Med. Journal,' vol. vii, 1786:—"An intimate friend of mine, a surgeon of great abilities, proposed to cut the ends of the ligatures close, and thus leave them to themselves. By following this plan we have seen stumps healed in the course of ten days. The short ligature, thus left in, commonly made its way out by a small opening, in a short time, without any trouble, or the patient being sensible of pain."

[3] 'Med.-Chir. Trans.,' vol. vi, 1814.

[4] *Catgut was first used on account of its absorbable qualities* by Sir A. Cooper. See 'Surgical Essays,' by Sir A. Cooper and Benj. Travers, vol. i, p. 125. A man, aged 80, with popliteal aneurism; ligature of femoral artery with catgut; ends cut short; wound healed by first intention in four days; patient up and about in three weeks. He remarks, "I confess that this case gave me much pleasure; the great age of the patient, the simplicity of the operation, the absence of constitutional irritation and consequently of danger, and his rapid recovery, lead me to hope that the operation for aneurism may become, at some future period, infinitely more simple than it has been rendered to the present moment" (ib., p. 129). Prof. Physick used buckskin in 1814 as an absorbable ligature.

cut short. He tried to get the ligature absorbed. His first case was a brilliant success, but his second case did not do well and he abandoned the practice. It is true that Galen[1] had long before recommended catgut, but he only did so if hemp or silk was not obtainable, and he says that the substance of the ligature should be such that it will not readily dissolve. To Lister[2] (1881) we are indebted for a method of preparing catgut which avoids the risk of its being absorbed too soon, and so makes it trustworthy.

The recognised practice at the present time may be said to be the use of the aseptic silk or catgut ligature so applied as to cut the coats of the vessel.

Lastly, it will be in the recollection of the Fellows that Mr. Barwell[3] (1879) has recently brought before the Society his plan of using tape-shaped animal ligatures for the ligation of arteries for the cure of aneurism. In his hands the practical application of this method has been most successful. Very recently Mr. Bennett May[4] has tied the innominate artery for subclavian aneurism with a ligature composed of six strands of catgut. The latter was

[1] Galen ('Methodus medendi,' liber xiii, ch. 22), speaking about bleeding, says, "But if, on laying bare the vessel, it should appear to you large, and to pulsate strongly, it is safer for the operator to put a (double) loop round it and to divide between; and let these ligatures be of a material that will not readily decompose. Such a material in Rome can be got from the Gaietans, who bring it from the country of the Kelts and sell it in the Via Sacra, which leads from the Temple of Roma to the markets. This is the easiest thing to get in Rome, for it is sold very cheaply there; but if you are practising your art in another city prepare for yourself some of the threads known as silk; rich women have these in many parts of the Roman empire, and especially in the large cities. If you cannot get this, choose the material least liable to decompose from among those that you can get where you are, such as fine catgut, for materials which easily decompose fall quickly out of the vessels, but we wish the knot only to fall out when the vessels have been well covered round with flesh, for the flesh which grows up in the parts of the vessels which has been cut off acts as a covering and stops its mouth, and when that has happened is the time for ligatures to separate without danger."

[2] 'Lancet, vol. i, 1881, p. 201.

[3] 'Med.-Chir. Trans.,' vol. lxii, 1879.

[4] 'Lancet,' vol. i, 1886, p. 1064.

drawn sufficiently tight to arrest all pulsation in the tumour, but not so tight as to impair the integrity of the arterial wall. There are few surgeons of the present day who practise the gentle treatment of the wall of the vessel, but to-night we desire to support their position from the experimental stand-point, and to recommend the employment of the small round absorbable ligature.

III.—*Opinion of the Present Day.*

The statement occurs or is implied in the language made use of in all recent text-books of surgery, that in the operation of ligature of an artery in its continuity the aim of the surgeon should be the complete division of the internal and middle coats of the vessel; and further, many and diverse ill results, such as hæmorrhage, or return of pulsation in the sac of the aneurism, are foretold as the probable consequence of any failure on the surgeon's part in carrying out this cardinal rule.

It is only necessary to refer to current surgical literature under the head of "Directions for the Operation," and whether the work of Bryant,[1] Erichsen,[2] Farabeuf,[3] Heineke,[4] Holmes,[5] or Mac Cormac[6] be consulted, the operator is told alike by each and all to tie the ligature strongly and steadily in order to divide the internal and middle arterial tunics. In most books, however, there is to be found evidence of considerable hesitation in the discussion of the subject. Heineke[4] is very uncertain, not knowing to which view to give the preference; he says, "It is only necessary that the artery be tied so tightly that the folds of the intima come in contact, but the ligature may

[1] 'Practice of Surgery,' 3rd edit., vol. i, p. 413.
[2] 'Science and Art of Surgery,' 9th edit., vol. i, p. 415.
[3] 'Manuel Opératoire,' 1881, pp. 24, 25.
[4] Billroth und Leücke, 'Deutsche Chirurgie,' Band 18, p. 94.
[5] Holmes, 'System of Surgery,' 3rd edit., vol. iii, p. 101.
'Surgical Operations,' Part I, 1885, page 19.

without disadvantage be drawn more tightly, in which case the inner coats are generally ruptured." Holmes and Erichsen give facts and arguments bearing on both aspects of the question. The former[1] says, "I have used Mr. Barwell's ligature myself with great success;" and again, "It is therefore probable enough that Mr. Barwell's view may be correct, but it cannot be said to be proved as yet, and I confess that I have always felt safer in drawing the ligature as tight as possible." Mr. Erichsen,[2] after mentioning the great danger of hæmorrhage subsequent to ligature of the first and second parts of the subclavian artery, concludes with the remark, "that the operation ought to be banished from surgical practice unless further experience shows that absorbable ligatures can be applied with certainty in such a way as to occlude the artery without division of its coats." Mr. Bryant[3] observes, when discussing the sloughing away of the portion of an artery included in a silk ligature, "that herein lies the weakness of the treatment by ligature." Lastly, Sir W. Mac Cormac[4] makes the following statements, which are germane to the object of this paper: "With some surgeons it is even now a question, as it was in Scarpa's day, whether or no it is desirable or necessary to divide by the ligature the internal and middle coats;" and again, "This practice has probably a better chance of success now than formerly as absorbable material is used."

IV.—*Authors' first Views.*

It is some years ago now that we first privately discussed the question of the ligature of an artery in continuity. The experiments of Scarpa and his contemporaries, and also those of the younger Cline and South[5]

[1] Loc. cit., 3rd edit., vol. iii, p. 101.

[2] Loc. cit., 9th edit., vol. ii, p. 201.

[3] Loc. cit., vol. i, p. 451.

[4] Loc. cit., p. 28.

[5] 'Chelius' Surgery,' translated by South, 1847, vol. ii, p. 221:—"A thread

(which show that by applying a ligature quite loosely around the carotid of a large dog the vessel becomes permanently occluded), seem to indicate that, by division of the coats of a vessel when not absolutely necessary to attain the end in view, surgeons are departing from that salutary law which precludes during operative measures any unnecessary injury to the tissues of the body. The evidence in this direction has gradually accumulated, and has led to the belief that the importance attached to damaging the arterial wall has been exaggerated and misstated, and that the operation of ligation in continuity ought to be reviewed in the light of recent advances in surgery and pathology.

V.—*Experimental Investigations.*

By the kind permission of Prof. Birch Hirschfeld and Dr. Hüber we put our views to the test of experiment in the pathological laboratory of the University of Leipzig. The experiments were made on sheep and horses, and we ligatured altogether sixteen carotids in sheep and three in horses. Strict antiseptic precautions were adopted; corrosive sublimate and carbolic acid being used for this purpose. The former answered best. The ligatures employed were kangaroo tendon from one twentieth to one twelfth of an inch in width, chromic catgut Nos. 3 and 4, and the green sulphurous catgut about No. 3 size. Except in Experiments 5, 6, 15, 16, 18 and 19 the ligature was drawn upon until pulsation on the distal side was arrested. The cavity of the artery is completely blocked in Specimens No. 15 and No. 19. It is much encroached upon in artery No. 18, but is scarcely involved at all in Specimens Nos. 5, 6, and 16. Excluding the above exceptions the

applied around the carotid artery of a dog so loose as not to interfere with the passage of the blood, is sufficient to cause inflammation, which will block it up completely, as was proved by an experiment made by my able master the younger Cline, and which I myself have repeated with the like result."

vessels were tied so that the lumina were nearly or wholly obliterated without any injury to the walls of the vessels. All the wounds in the sheep healed by first intention and remained aseptic throughout. Those in the horses suppurated more or less. The animals were killed at such periods as to allow of the vessels being removed at times varying from nine hours to seventy-three days. It will be observed that most of the vessels were removed from the bodies of the animals within three weeks. It was desired first to demonstrate the action of the small round ligature in occluding a vessel without damage to its wall, and to show that such an operation was easy and practicable. If a longer period had been selected it would have been difficult to convince everyone that the walls of the vessels were not ruptured, because the plastic process after a time obliterates the normal outline and the usual landmarks. Having proved the ease with which, by the small round ligature, ligation in continuity without rupture of the tunics can be done, we hope at some future time to make further experiments of a like kind, but with the arteries removed from the bodies of the animals at longer periods after ligature. Experiment 19 illustrates this point, but at present it stands alone. The carotid of a horse is seen permanently occluded on the fifty-first day.

Experiments 5, 6, and 16 taken together are very important. In No. 6 the artery is contracted and pervious after seventy-three days. In No. 5 (fifty-eight days) and in No. 6 (forty-four days) the vessels are filled with clot which is not adherent to the wall and which shows no evidence of organising changes. In each of these cases the vessel was scarcely, if at all, constricted by the ligature, and the tunica intima was thickened on account of its proximity to the clot. In all three a coagulum had formed which in one case had been washed away, whilst in the other two it would soon have met with the same fate. We can conclude therefore from these three experiments :—

That South and Cline were mistaken when they stated that an artery became permanently occluded by having a

ligature placed loosely around it; though a coagulum does form which lasts for about sixty days.

The kangaroo tendon was tied with the reef-knot, the catgut with the "double hitch" or surgical knot.

The majority of the vessels were immersed for preservation in equal parts of glycerine and absolute alcohol and brought to England for further examination, but some (six) were placed in carbolic solution (1-20). The alcohol caused the vessels to shrink to about a quarter of their original size.

Each vessel was split longitudinally through the middle of the knot of the ligature, so that the portion of the arterial wall subjacent to the knot and most exposed to injury comes well into view. One half was saved to be mounted as a naked-eye specimen in glycerine jelly, and the other part was reserved for the microscope.

We have much pleasure in thanking Mr. Horsley for his kindness in allowing us to use the Brown Institution for the purpose of working up our material.

VI.—*Specimens described and considered.*

Scheme of Experiments.—The following carotids of sheep were tied with kangaroo tendon. The ligature was applied except in the two cases mentioned below, so as to arrest the current of blood.

Exp. 1.—Carotid seven days after ligature. Lumen not quite obliterated. Commencing organisation of new material which is taking the place of the clot.

Exp. 2.—Carotid ten days after ligature. Lumen not quite obliterated. Organisation in clot more evident.

Exp. 3.—Vessel fourteen days after operation. Lumen occluded. Increasing development of new material in coagulum.

Exp. 4.—Vessel twenty-one days after operation. Lumen nearly occluded. Near the ligature the organisation of plastic material extends across the clot joining the opposite intimæ. (See Plate XI.)

Exp. 5.—Carotid fifty-eight days after operation. The ligature was placed loosely around the vessel without any attempt being made to control the passage of blood through it. The endothelial lining of the innermost coat is much thickened. The surface of the ligature is commencing to give way before the attack of the leucocytes. A clot fills the vessel which is not adherent, in which no organisation is taking place, and which would have been washed away in the blood stream if the animal had been allowed to live. This point is illustrated by the next experiment.

Exp. 6.—Carotid seventy-three days after ligature. As in the last case so in this, the ligature was applied around the vessel without any attempt being made to control the passage of blood through it. The ligature can still be seen with the naked eye. There is evidence that it slightly constricted the arterial wall. With the microscope its outline appears irregular; this is caused by absorption by the cellular invasion. In a very short time more, without doubt, it would have entirely disappeared. The vessel itself is contracted and diminished in size but pervious. The internal tunic is much thickened, especially the endothelial layer. It is certain that it was for some time obstructed by a coagulum which has been carried away by the blood stream.

The following carotids of sheep were tied with catgut, Macfarlan's No. 3 chromic catgut was employed except in the instances detailed. Each ligature (except in Experiments 15 and 16) was intentionally pulled upon until on its distal side the pulsation in the artery had ceased.

Exp. 7.—Vessel nine and a half hours after operation. Chromic catgut No. 4 was the ligature used. Lumen obliterated by the ligature.

Exp. 8.—Carotid twenty-four hours after operation. Green sulpho-chromic catgut No. 3 was used. Lumen occluded.

Exp. 9.—Vessel three days after operation. Calibre obliterated by the ligature.

Exp. 10.—Vessel seven days after operation. Lumen

not quite obliterated. Commencing organisation of clot near seat of ligature.

Exp. 11.—Vessel nine days after ligature. Lumen nearly occluded.

Exp. 12.—Vessel ten days after ligature. Calibre nearly obliterated.

Exp. 13.—Carotid fourteen days after operation. Calibre obliterated. Progressive organisation in clot.

Exp. 14.—Vessel twenty-one days after ligature. Calibre obliterated by ligature. Extensive organisation of plastic material in clot near the seat of ligation.

Exp. 15.—Vessel thirteen days after ligature. Complete obliteration by the ligature of the lumen of the vessel.

Exp. 16.—Vessel forty-four days after ligation. No attempt was made in this case to arrest by the ligature the passage of blood through the artery. A coagulum is present which is not adherent and which in the specimen has mostly fallen out. It shows no evidence of vital changes, and if the animal had been allowed to live would without doubt have been carried away in the blood stream. The tunica intima is much thickened. The catgut ligature is still holding its own. It must have been exceptionally well prepared to resist absorption for so long. The leucocytes, however, are working their way in from the surface, but yet the ligature would probably have remained unabsorbed for another fortnight if the sheep had been allowed to live.

The following carotids of horses were ligatured:

Exp. 17.—Vessel ten and a half days after ligature with kangaroo tendon. Lumen not quite obliterated. Commencing organisation in clot at the seat of ligation.

Exp. 18.—Carotid fourteen days after operation. Catgut Macfarlan's No. 3 was used. No attempt was made to completely arrest the flow of blood at the ligatured point. The lumen is encroached upon but not nearly obliterated by the ligature. In the specimen the clot has dropped out except at the point of ligation. Much sup-

puration took place, hence the great amount of plastic exudation. The ligature is being rapidly absorbed.

Exp. 19.—Carotid fifty-one days after operation. Chromic catgut No. 3 was the ligature used. The calibre at the ligatured point was evidently not quite obliterated. Organisation in the clot in the neighbourhood of the ligature is complete, for a fibrous union extends across the interval which had previously been occupied by coagulum from the inner coat of one side to the inner coat of the opposite side. The ligature is absorbed. No trace of it is visible.

The macroscopic[1] and microscopic examination of the specimens show:

1. That in no instance were the arterial coats injured by the ligature.

2. That except in three cases (Experiments 5, 6, and 16), in which the arteries were only slightly constricted, the lumina of the vessels were either wholly or nearly occluded. In other words, at the point of ligature either the internal coat of one side was in apposition with the internal coat of the opposite side, or a thin strand of clot blocked the lumen of the tube at the point of constriction and was continuous with the main body of the clot both above and below.

3. That external to the artery, surrounding the ligature and extending a short distance on either side of it, was a small amount of constructive exudation-material, due to the presence of the ligature and the disturbance of parts which was a necessary coincidence of the operation. When suppuration took place, as in Experiment 18, the amount of plastic exudation thrown out was much greater.

4. That the ligature, whether of tendon or catgut, to the naked eye is practically unaltered, is not producing any irritation, and is holding well at the end of twenty-one

[1] The macroscopic specimens are preserved in the museum of the Royal College of Surgeons.

days. In Experiment 19, fifty-one days after operation, the catgut ligature has disappeared. In Experiment 6, seventy-three days after operation, the tendon ligature is almost entirely dissolved.

5. The gradual diminution and contraction of the vessel, which was most marked on the proximal side of the ligature. (Those arteries which were taken from the bodies of the animals twenty-one days after operation, were discovered by measurements taken immediately after death to have shrunk to less than half their diameters at the time of ligature.)

6. The decolorisation and absorption of the clot and the organisation of plastic material which is taking its place, is well seen in the neighbourhood of the ligature when the latter wholly or nearly obstructs the cavity of the vessel, and in three weeks by this process the proliferating endothelium of one side is in vital union with the proliferating intima of the opposite side—the clot space being thus rapidly bridged across. When the vessel is only slightly constricted a coagulum forms but it remains a "foreign body" destitute of vital action until it is carried away by the blood-stream.

7. A careful investigation of this series of experiments demonstrates clearly—

1. That when an artery is only slightly constricted it becomes temporarily blocked for a considerable time—from fifty to seventy days. It then, much diminished in size, resumes its function as a carrier of blood.

2. That when an artery is wholly or nearly occluded by the ligature, plastic processes (which can be readily traced from their commencement a few hours after ligation to their completion fifty days later in the microscopic sections) supervene which permanently block the lumen of the vessel, which unite the inner coats of opposite sides and which practically finally convert the artery at the seat of ligature into a solid fibrous band.

VII. *The Coagulum.*

The clot which forms above and below the seat of ligature is not in any sense a necessary part of the process by which an artery is obliterated. Ziegler,[1] Cornil and Ranvier[2] and others[3] have advocated this view. Travers[4] experimentally proved its non-essential nature though many before his day had combatted the doctrine of Petit.[5] The coagulum within a deligated vessel is as much a foreign and dead substance as the clot on the flap of an amputation stump. It, like the aseptic animal ligature encircling the artery, is gradually absorbed. Its function is to act as a barrier or buffer between the impulse of the blood-stream and the seat of ligature where the important plastic actions are in progress which might otherwise be disturbed or interrupted; and also, perhaps, in the vicinity of the ligatured point to afford some support—ladder-like—to the plastic[6] effusion as the latter climbs across the cavity of the vessel. Whether the tunics be ruptured or not, the coagulum takes no part in the adhesive process by which the final occlusion of the vessel is secured, but upon its deficient formation, when the arterial wall is damaged, may depend an attack of secondary hæmorrhage, especially if a large collateral branch be close to the deligated point. The adhesive changes only involve the clot in the immediate neighbourhood of the ligature. The remainder of the clot, which is the greater part, after some time disappears and its place is occupied with fluid blood again. These changes always end in a permanent diminution of

[1] Ziegler, 'Pathological Anatomy,' pt. 2, par. 255.

[2] Cornil et Ranvier, 'Manuel d'Histol. Pathol.,' 1881, vol. i, p. 601.

[3] Spence, 'Lectures on Surgery,' p. 515; Farabeuf, 'Manuel Opératoire,' 1881, p. 28.

[4] Loc. cit.

[5] Petit (1710) was the first to conduct experiments on the ligature of arteries. He thought the coagulum was the chief factor in the arrestment of hæmorrhage and the process of obliteration.

[6] John Bell, 'Principles of Surgery,' 1801, vol. i; Jones, loc. cit., p. 160.

the calibre of the vessel above and below the ligatured point.

Our specimens illustrate these views. In each case in which the vessel cavity was entirely or almost entirely obliterated by the ligature, the microscope shows that a cellular infiltration is taking place into the wall of the vessel around the ligature and into the clot. The longer the interval which elapsed between ligation and the death of the animal so much the more organised is the plastic effusion. By means of these cells the clot near the ligature is decolorised and with the ligature is at last completely absorbed. Vessels developed from the formative material which is taking the place of the clot are to be seen at the end of a fortnight or three weeks passing across from the proliferating intima, or plastic effusion, within the intima of one side to the intima of the opposite side. In other words the inner coats of opposite walls are commencing to be adherent. Already there is a living connection, and the intervening space is filled with a tissue which only requires a short time more for its perfect development.

The same adherent changes are evident in the thin strand of clot, which in some instances is visible at the point of ligation, as are seen in the main body of the clot just above and below the ligature when the lumen of the vessel is obliterated. In those sections in which the tunica intima of one side is shown approximated to the tunica intima of the other, there is a direct vital adherence without the assistance of any intervening material, but dependent upon, as in the former case, the plastic cellular extravasation. This is of the utmost importance, for it proves that it is of comparatively little moment whether the ligature which does not damage the coats completely or almost completely closes the lumen of the vessel. In Experiment 19 the carotid of a horse which was not completely closed by the ligature is converted at the seat of constriction into a solid fibrous band.

Reidel[1] is said to have made the inner surfaces of an

[1] Quoted by Ziegler, 'Pathol. Anat.' (Eng. trans.), vol. ii, p. 14.

artery cohere by multiplication of the opposed endothelial cells without the formation of clot. Of the accuracy of this observation we have grave doubts. Any alteration in the endothelial lining would certainly lead to the formation of a coagulum, and the endothelial multiplication in our specimens is always accompanied by a leucocytic extravasation, the latter apparently being of more importance than the former. A long time after ligation the clot disappears and leaves the interior of the vessel—on either side of the ligatured point—in shape something like a hollow cone. This may possibly be the explanation of the statement of Reidel.

The time at which the clotting took place in these experiments is a matter of doubt, but it probably supervened soon after the operations were over. The shortest time which elapsed between ligation and the death of an animal was nine hours. Here the clot was perfectly formed. When the coats are ruptured the clotting commences at once on the infolded edges of the cut tunics, but in these arteries no such cause was present and the development of the coagulum must be attributed, not to the stasis of the blood current, but to the change in the vital state of the arterial wall due to the pouring out of lymph at the point of ligature.

VIII.—*The Ligature.*

Experiments upon arteries in the post-mortem room show:

1. That the effects produced by the use of a silk or other ligature applied in the ordinary way are not uniform. The middle coat is sometimes only partly cut through but in other instances a mere strand of tissue representing the outer coat alone remains, which remnant is thinnest and quite transparent under the knot of the ligature. Here then is ample reason for the occasional occurrence of

hæmorrhage; for not only is the wall of the vessel nearly divided at every point but in the knot region the outer tunic has almost given way. During last autumn the opportunity was afforded of dissecting a case in which secondary hæmorrhage, preceded by some suppuration, occurred after ligature of the carotid. The wall of the vessel had given way at its weakened and most disabled point, *i. e.* nearest the skin under the knot.

2. That there is no difficulty in tying an artery with a small round ligature sufficiently tight to make it impermeable to water without the least damage to the coats. The occlusion is caused by a longitudinal wrinkling of the wall.

3. That a tape-shaped animal ligature a quarter of an inch in width will rupture the coats of an artery if force be used. The knot of such a ligature is clumsy and the tape does not lie flat in the neighbourhood of the knot.

The choice of material for the ligature has been much debated.

1. It is not easy to make silk aseptic. This is probably on account of the presence of recesses in which bacteria can lodge. When employed outside the peritoneum there is some uncertainty as to what will happen to it. It may become encysted or it may ulcerate out. The question in the past has been one of gangrene at the seat of ligature and of inflammation on either side; now suppuration in such a wound is and ought to be the exception. Senn[1] has shown that a silk ligature applied to an artery in its continuity without damaging the tunics always cuts its way through the vessel and frequently becomes encysted by the side of it. Dr. Kölliker, of Leipzig, informed us that of eighty amputations antiseptically performed in which he had carefully searched for the silk ligature used for the main vessel, he had only succeeded in finding it in about one third of the cases. He supposed that some of the ligatures had escaped his observation and that it was fair to estimate that one half remained encapsuled in

[1] 'Trans. Amer. Surg. Assoc.,' vol. ii, 1885, p. 345.

the stumps and one half came away in the discharges. Silk[1] then hardly fulfils our ideal of a perfect ligature for aseptic wounds.

2. Of late years catgut has been extensively used. Lister[2] lays stress upon its preparation, specially insisting upon the scraping off of the mucous and peritoneal surfaces of the bowel from which it is made; and upon the fact that it is absorbed from the surface. Our microscopic preparations demonstrate, however, that our catgut had not had the mucous coat removed for villi and mucous follicles are to be seen. To the naked eye there is no change visible even at the end of twenty-one days. With the microscope at the end of the third day the dendritiform arrangement of villi and mucous follicles can be easily made out following certain wavy lines which cross the section of the catgut. On the seventh day after ligature this appearance is not nearly so clearly defined, and there are some cracks or splits running from the surface towards the centre. Along these splits leucocytes are gathering. Fourteen days after operation the dendritiform picture has disappeared and the cracks are wider and deeper; and at the end of twenty-one days the fissures are still more marked and the circumference of the catgut and the sides of the cracks bear evidence at several points of the eroding and absorbing influence of the surrounding leucocytes. (Plate XIII.)

In Experiment 18, in which profuse suppuration followed the ligature of the carotid of a horse, the ligature—catgut—is breaking up rapidly at the end of fourteen days. In Experiment 19, in which the vessel was removed fifty-one days after operation, there is no trace of the ligature. In the microscopic sections of artery No. 16 the catgut, which is apparently exceptionally good, is holding well after forty-four days. We think that well prepared chromic catgut will last for one month or more; and, as a ligature upon an artery in continuity, will not give

[1] Holmes, 'Surgery, its Principles and Practice,' p. 94.

[2] Loc. cit.

way in a less time unless very profuse suppuration occur.

3. The tendon used by us has several points in its favour:

a. The structure is continuous throughout, and there are no spaces as there are in catgut, due to twisting in its preparation.

b. It does not split or crack during absorption, which takes place "from the surface."

c. It is easily made aseptic.

d. It is only gradually, and after a long time, acted upon by the living materials which encompass it.

Kangaroo tendon is very convenient for practical use, being strong, of ample length, and becomes as supple as silk by soaking for half an hour in tepid sublimate solution.

The tendon ligature shows scarcely any absorption on the surface at the end of twenty-one days. Leucocytes are collecting, as in the catgut specimens, in a dense mass on the outer side. In Experiment No. 5 the tendon is seen fifty-eight days after operation and does not exhibit much change except on the surface microscopically. In Specimen No. 6 the tendon seventy-three days after ligature shows unmistakeable signs of disappearance. The circumference is deeply indented and wavy in outline.

We consider that kangaroo tendon ligature may be looked upon as trustworthy for at least two months.

IX. *Objections discussed.*

The following are some of the objections which may be raised:

1. *That conclusions based upon the ligature of the carotids of sheep, and which are intended to be a guide to practice, are founded upon an analogy which is not wholly supported by the facts,* because:

A. The circulation in sheep and other herbivora is not so vigorous as in man.[1]

B. The carotid of a sheep is not quite so large as a human carotid.

To meet this objection the carotid artery of the horse was ligatured in three instances. This vessel is much larger and the blood pressure is much greater than in the corresponding artery of man. The macroscopic and microscopic preparations of these three horse carotids show exactly the same changes as are seen in the ligatured carotids of sheep; and in Experiment 19 the carotid of a horse at the end of the fifty-first day is converted at the ligatured point into a solid fibrous mass.

2. *That it does not matter under the Listerian system whether the tunics be ruptured or not; that there is no danger involved in the division of the coats, and that the result cannot be (with primary union of the wound) disastrous to the patient.*

There can be no dispute about the supreme desirability of obtaining perfect asepsis, but to the belief as stated above we cannot subscribe, because:

A. It is not justifiable to do more than is absolutely necessary to attain the end in view.

B. It cannot be expected that wounds will always heal by first intention and remain aseptic throughout. Though most cases of ligature of arteries in their continuity with strict antiseptic precautions are successful, it is not well

[1] The relative blood-pressure in the carotid of man, compared with that in the same vessel of other large mammals, is as follows:

Horse	160—220 mm. of mercury.
Sheep	155—210 mm. „
Man	150—200 mm. „
Large dog	140—180 mm. „

From private letter (Mr. Langley, of Cambridge).

The relative size of the dead carotid of man, compared with the same vessel of the horse and sheep, is as follows:

	Outside diameter.	Inside diameter.	Thickness of coat.
Horse . .	12 mm. ...	9 mm. ...	1½ mm.
Man . . .	7 mm. ...	5 mm. ...	1 mm.
Sheep . .	5½ mm. ...	4 mm. ...	¾ mm.

to trust too much to asepsis. It has already been shown what may happen if asepsis be not perfect.

The minimum of unsuccessful cases may probably be greatly reduced by the employment of means which, while efficiently occluding the vessel, do not at the most critical moment, and at the situation of greatest strain, destroy the strength of the arterial wall.

3. *That it is more difficult to tie a vessel without damaging its coats than to tie it in the ordinary way.*—To this statement a denial must be given, for we are sure from experiments upon dead arteries that it is just as easy to learn thus to tie an artery as to ligature one by main force.[1] It is always possible to tell at once when the ligature must not be drawn any tighter, for a certain resistance is felt by the fingers which, if overcome, is overcome suddenly and with a snap, and means the giving way of the two inner coats of the vessel; and further, the cessation of pulsation in the artery or its branches beyond the ligatured point, or in the case of aneurism the cessation of pulsation of the tumour, is an important indication to the operator to abstain from tightening much more the knot of the ligature.

4. *That it is not easy perfectly to occlude an artery without rupturing its coats.*—This, however, is not the fact. It is quite easy in the post-mortem room to tie an artery with an ordinary silk ligature without any damage to the tunics and yet so completely to occlude the vessel as to prevent the passage of any water even when the latter is forced in by means of a syringe. The specimens show moreover that it is not necessary that the tunica intima of one side should be in apposition with the tunica intima of the opposite side, though in some instances this perfect approximation does obtain. Supposing the lumen of the artery not to be completely closed by the ligature and a small space to remain through which blood could find its way in small quantity, clotting must inevitably soon take place. But even if coagulation were delayed for some

[1] Farabeuf, loc. cit., p. 26.

hours the trickling of a little blood through the vessel at the ligatured point would be by no means disadvantageous from the point of view of the formation of a firm clot in the sac of the aneurism.

5. *That the ligature may rapidly dissolve so that the circulation through the vessel becomes quickly re-established.*—This has happened in actual practice[1] with carbolic catgut. Such a result is not surprising, considering that ligatures of badly prepared catgut may separate and be found in the discharges thirty-six or forty-eight hours after an operation.

Our specimens show that properly prepared chromic catgut or kangaroo tendon possesses great powers of resistance to the action of living tissues and prove therefore that with well-selected materials an untoward event of this sort could not happen.

6. *That the vessels may become pervious after a more or less lengthened period by absorption of the ligature and canalisation of the clot or new material at the point of ligature.*—To this objection it may be urged:

A. That aseptic ligatures can only be absorbed or encapsuled. That the former would certainly have happened in our cases but that the materials used would have been entirely absorbed, only after some months when all surrounding parts would have changed into fibrous tissue.

B. That though a clot, when it remains at the point of ligature simply as a lifeless mass (as in those instances in which the arterial wall is only slightly constricted) must be ultimately carried away in the blood stream, yet when organisation does occur to the extent of bridging over the interval occupied by the coagulum, it must continue until the "new material" is changed into a permanent fibrous mass.

C. That granting for the sake of argument that the circulation would be re-established in some modified degree, it is obvious that such an event could not occur

[1] Bryant, 'Surgery,' 3rd edit., vol i, p. 414; Treves 'Brit. Med. Journ.,' vol. i, 1881, p. 232.

except after the lapse of many weeks, and that supposing *e. g.* that the operation was performed for the cure of aneurism, the re-establishment of the circulation would be heralded long before by the effectual cure of the disease as far as the cure was dependent upon the passage of blood through the vessel tied.

7. *That if suppuration occur in the wound the patient would be placed in a position of greater danger than if the arterial wall had been dealt with in the usual way.*—We are, however, convinced from the study of the history of ligature before and since the antiseptic era, that the danger to the patient is greatly augmented by the division of the two internal layers of the arterial wall. We have dissected a case in which the popliteal artery passed safely through the centre of a large abscess cavity, suffering only a slight thickening of its sheath and outer coat, and had there been any artificial injury to the barrier of the arterial wall the chances of a disastrous termination from hæmorrhage would have been very much magnified. In St. Thomas's Hospital museum are the carotids of horses tied with rupture of the tunics by Travers. In several of these cases severe secondary hæmorrhage occurred, in one case to syncope. On looking at our three specimens it will be seen that hæmorrhage could not occur, for the vessel wall in each case is intact, though suppuration supervened, and in Experiment 18 was most profuse. The strongest section of the arterial wall, when the coats are uninjured, is at that point where it is strengthened by a scaffolding of ligature plus the sheath of plastic exudation material which is rapidly developed into young fibrous tissue.

8. *That*[1] *plastic lymph is effused as a consequence of the injury done to the coats, and upon the amount and vitality of the effusion depends the safe closure of the vessel. That*[2] *the injury done to the intima is of cardinal importance for the formation of thrombus and the development of adhesive*

[1] Mac Cormac, loc. cit., p. 25.

[2] Ib., p. 29.

inflammation. That[1] *if these coats are not lacerated it is probable that no lymph will unite their opposed surfaces.*— The naked-eye and microscopic preparations of the vessels in our experiments, however, show an effusion of lymph which is ample for the purpose in view, viz. the occlusion of the vessel, so that the plastic exudation cannot be said to be dependent in quality, though possibly in quantity, upon rupture of the tunics.

9. A.—*That when two endothelial surfaces are brought into contact they unite with difficulty, and that therefore it is necessary to interrupt the continuity of the tunics.*

B.—*That it is an advantage to bring, by means of the cutting ligature, the adventitia of one side into close relation with that of the opposite side, because union between areolar structures is rapidly effected.*

Our preparations clearly demonstrate that these are theoretical issues having no foundation, and that union is obtained as firmly and as rapidly, and more safely, when the tunics are undamaged than when they are divided. Other endothelial surfaces when in contact are known to adhere on the least provocation. Ziegler[2] says "that a blood-vessel has an anatomical analogy to the serous cavities" and that "the process by which a thrombus is organised resembles most closely the plastic inflammation of a serous membrane." The presence of a ligature even when loosely applied round an artery is sufficient to cause a slight deviation from the normal nutrition of the part, accompanied by plastic effusion, proliferation of the endothelium, and coagulation of the blood.

Ziegler figures an organising thrombus from the femoral artery of an old man. The tunics had been ruptured and the examination was made three weeks after ligature. Let this picture be compared with the process as seen in a sheep's carotid twenty-one days after operation without division of the coats. In the latter case the process of organisation is much more advanced than in the former,

[1] Holmes's 'System of Surgery,' 3rd edit., vol. iii, p. 101.

[2] Loc. cit., p. 11.

for in the human femoral the blood-cells of the clot are visible and the large fusiform and ramified cells are only beginning to be formed near the endothelium and to extend inwards between the cells of the coagulum; but in the sheep's carotid a network of these formative cells has already extended from the inner tunic of one side across the clot to the inner tunic of the opposite side, and the individual cells of the coagulum cannot be distinguished. In other words, the constructive process as seen in the plastic effusion, proliferation of the endothelium and disappearance and absorption of the blood-cells and fibrin of the clot may be said to progress at any rate as rapidly when the integrity of the arterial wall is secured as when it is destroyed.

X. *Conclusions.*

The conclusions at which we have arrived may be briefly stated as follows :—

1. That the operation of ligature of a large artery in its continuity should be performed without damage to its wall.

2. That the rupture of the coats of an artery during ligation in continuity is a useless and dangerous proceeding. Useless because the surgeon can secure the effectual attainment of his object, viz. the occlusion of the vessel, by a measure at once safer and less severe; and dangerous on account of the possible occurrence of some untoward event, such as hæmorrhage or secondary aneurism at the seat of ligature, which could not happen if the wall of the vessel were uninjured by the ligature.

3. That if the wall of the artery be diseased, the advantages attending ligation without rupture of the tunics are much magnified. It sometimes happens that the surgeon on cutting down upon a large artery observes a state of atheroma so extensive that he is obliged to close the wound and ligate a vessel nearer the heart and thus expose his patient to considerably increased risk. There is no escape from such a dilemma under the system which

declares that the arterial coats must be divided; but with a non-irritating aseptic ligature so applied as not to lessen the power of the arterial wall but actually to be a source of additional strength to it where it is most desirable to conserve this quality, the question of ligation is seen under entirely new auspices, and the occlusion of a diseased artery would be undertaken with an assurance of success almost equal to that which obtains when a healthy vessel is in question.

4. That when the coats of an artery are uninjured by the ligature, the danger of ligation near a large collateral branch is wholly avoided, because—

A. No danger can accrue from hæmorrhage when the wall of the vessel is intact.

B. The formation of clot upon which the safety of the patient so much depends, if the wall of the vessel be damaged, has really nothing to do with the adhesive changes which take place in a ligatured vessel.

C. The plastic actions which proceed at the place of ligation are practically alike whether the tunics be ruptured or not. In the former case, however, any retardation of the constructive process, especially when in the vicinity of a large collateral branch (on account of the general condition of the patient or from accidental slight septicity of the wound) may be attended with grave risk to life—a risk which can by no means be made light of even when the course of events in the wound is apparently favorable. On the other hand when the tunics are undamaged the nearness of a collateral branch and suppuration in the wound are comparatively immaterial, and the reparative and adhesive efforts of nature as seen in the effusion and organisation of lymph develop, even when delayed, an additional stay to the unweakened and living arterial wall.

5. That the ligatures employed in this series of experiments were probably in all cases larger than was absolutely necessary to secure the obliteration of the vessels to which they were applied. Comparatively speaking they were not large. It would appear that a small round

aseptic ligature which will not become absorbed in a less time than three weeks, and which during that period holds firmly so as to cause a constriction of the arterial wall, and complete or almost complete obstruction of the cavity of the vessel will so influence the nutrition of the part that permanent occlusion will follow.

6. That it is no more necessary to use a flat tape-shaped ligature (as recently revived by Mr. Barwell for the purpose of preventing damage occurring to the arterial wall during ligation) than to rupture the coats of the vessel. The small round ligature is the most easy to manipulate, and it is not difficult to learn to apply it in the manner here indicated.

7. That the essentials to be observed in the ligature of arteries in their continuity are:

A. Complete antiseptic precautions to ensure the primary union of the wound.

B. A non-irritating aseptic ligature such as kangaroo tendon or chromic catgut, which will remain for a considerable period without becoming appreciably altered by the temperature and tissue environment of the living body.

C. The application of the ligature so as to close or almost close the lumen of the vessel without causing the least injury to the arterial wall.

The sum up, we venture, though fully conscious of the incompleteness of the experimental proof which is placed before the Society to-night, to advocate—

1st. The use of antiseptic precautions.

2nd. The employment of the small round absorbable ligature.

3rd. The maintenance of the integrity of the arterial wall.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' New Series, vol. ii p. 112).

DESCRIPTION OF PLATES XI, XII, AND XIII.

The Ligation of the Larger Arteries in their continuity: an Experimental Inquiry, by CHARLES A. BALLANCE, M.S., and WALTER EDMUNDS, M.C.)

PLATE XI.

The carotid of a sheep twenty-one days after being ligatured with kangaroo tendon.

FIG. 1.—Under low power, showing that the wall of the vessel is uninjured. The spot from which the high power drawing (fig. 2) was taken is marked by lines.

FIG. 2.—Section taken through the clot from one side of the vessel to the other in the immediate neighbourhood of the ligature. The cellular invasion and the proliferating endothelium are well seen. The blood-cells of the coagulum have become indistinguishable. The new material which is absorbing the clot, and taking its place, is already so far developed as to form a vital connection between the intimæ of opposite sides.

PLATE XII.

Carotid of a horse fifty-one days after being ligatured with chromic catgut.

FIG. 1.—The lumen of the vessel was, as far as can be made out, not quite obliterated by the ligature. There is no trace of the catgut to be discovered, even with the microscope. The place of the clot is taken by connective-tissue material, which has completely fused with the intimæ of opposite sides. Spot from which high power drawing (fig. 2) was taken is marked by lines. *Probable position of ligature.

FIG. 2.—High power drawing of part enclosed by lines in fig. 1. Complete fibrillation of new material which is taking the place of the clot, and fusion of new material with the wall of the vessel on either side. The organisation is more advanced nearer the ligature.

PLATE XIII.

FIG. 1.—Chromic catgut (No. 3) removed from a sheep three days after being used for tying the carotid. A dense mass of leucocytes is collecting on the outer side of the ligature. The mucous coat has not been removed in the manufacture of the ligature. The intestinal villi and crypts are clearly visible.

FIG. 2.—Showing rapid destruction of chromic catgut used for the ligation of the carotid of a horse fourteen days previously.

FIG. 3.—Chromic catgut ligature forty-four days after being employed for ligaturing a sheep's carotid. It is still holding, and likely to last for some time longer. This piece of catgut is exceptionally good; it was probably prepared with care.

FIG. 4.—The remains of a kangaroo tendon ligature seventy-three days after ligation of a sheep's carotid.

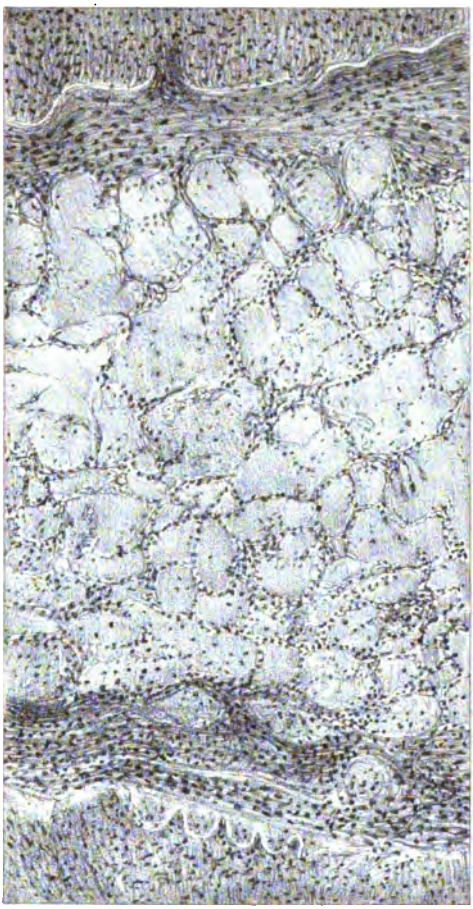

Fig. 2.

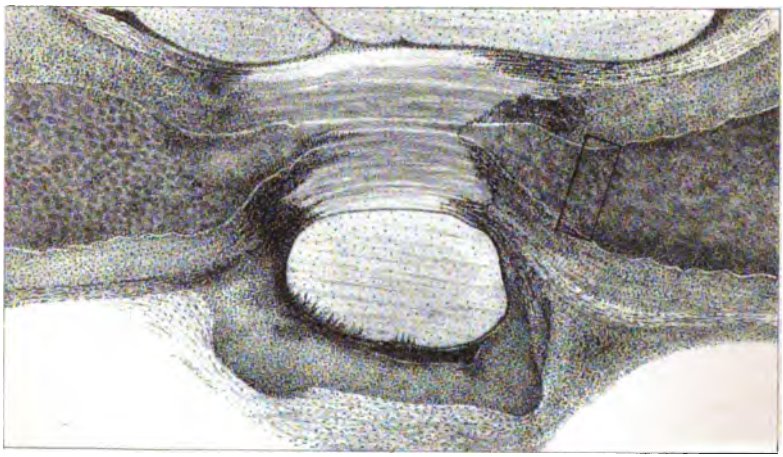

Fig. 1.

Mintern Bros. lith.

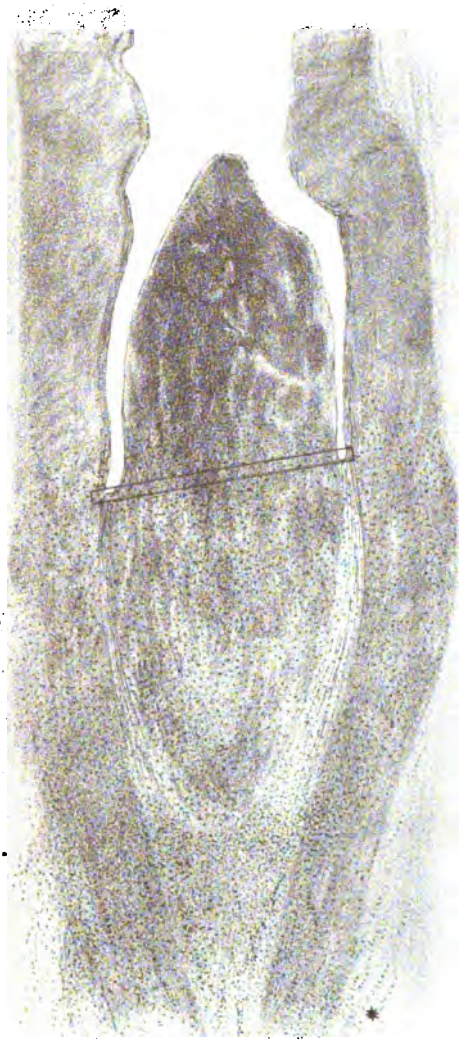

Fig. 1.

Fig. 2.

Mintern Bros. lith.

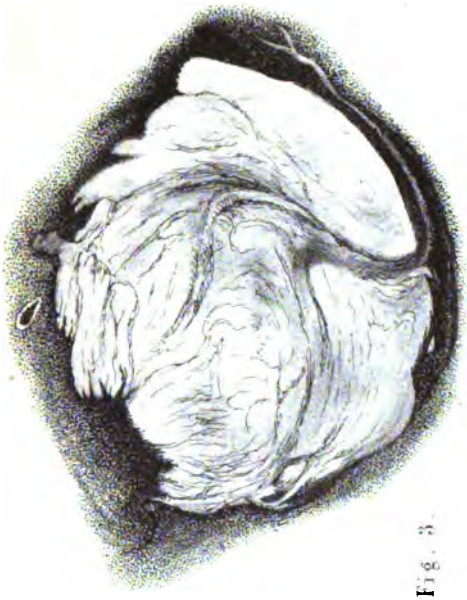

Fig. 3.

Fig. 4.

Fig. 1.

Fig. 2.

Muntern Bros lith.

CONGENITAL ABSENCE OF HAIR AND MAMMARY GLANDS

WITH

ATROPHIC CONDITION OF THE SKIN AND ITS APPENDAGES

IN

A BOY WHOSE MOTHER HAD BEEN ALMOST WHOLLY BALD FROM ALOPECIA AREATA FROM THE AGE OF SIX.

BY

JONATHAN HUTCHINSON, F.R.S., LL.D.

Received January 19th—Read May 11th, 1886.

THE subject of this case, a boy æt. 8½, presented a very peculiar withered or old-mannish look, all his features being thin and pinched. His fingers were shrivelled, and dusky, and their nails, which also were remarkably thin, were curved backwards so as to present more or less of hollow in the middle. His head was large and the anterior fontanelle not quite closed; the scalp was exceedingly thin, and with the exception of a quantity of down, was quite bald. It looked semi-transparent and tight, and the veins coursing in it were everywhere conspicuous. The veins were probably larger than natural. A large trunk came down the forehead on each side of the eyebrow and communicated by a transverse branch at the

root of the nose. The inosculations across the middle line of the scalp were many. There was a peculiar blue tinge about the lips; it involved the skin and not the prolabium only. At first I thought that this was due to accidental staining; but after he had been half an hour in my room it much diminished, as did also the turgescence of the veins of his scalp. His lips were exceedingly thin. His teeth were all cut and were tolerably regular, but his incisors did not stand quite straight, most of them had some slight inclination into the mouth. On his shoulders he was so thin that his coracoids and the outlines of his acromion processes could be easily seen; the skin over them being not much thicker than brown paper. The tightness of skin was nowhere very conspicuous excepting on the scalp; thus, on the abdomen, arms, and thighs the integument was quite loose but everywhere very thin. His muscular development was slight in all parts excepting the thighs, which felt hard and had muscles quite out of proportion to the rest of his body (this remark does not apply to the buttocks). His genitals presented a very remarkable contrast to the rest of his body. The parts about the pubes and upper part of the scrotum were so full and plump that a suggestion occurred that he must have double hernia. This, however, was not borne out by examination, and I believe the simple fact was that the scrotum and adjacent parts of skin were in the state of those of a normally stout child, whilst everywhere else the skin, subcutaneous cellular tissue, and panniculus adiposus were almost absent. The true scrotum was small, naturally corrugated, and occupied only the lowest part of the genital pouch which I have described. I do not think that there was anything very unusual in this state in a child, but must admit that possibly there was some excess of subcutaneous development about the pubes and root of penis. His testes were well placed and of normal size. His penis, except that there was phimosis, was quite natural. His toes and their nails were in the same condition as his fingers. He did not walk quite perfectly,

always keeping his knees a little bent, but I could not make out any definite muscular defect. One other remarkable feature remains to be mentioned, he had no nipples and their sites were occupied by little patches of scar. These scars were exceedingly superficial and slightly marked, but I am sure that they were there. Nothing like a mammary gland could be traced.

The history which the mother gave me of the child was that he had had no ailments since his birth, was of cheerful disposition, and very intelligent. It had been necessary from cross presentation to turn during delivery, and for some days after birth he had been very blue, probably in a state of partial cyanosis. He was still liable to vary very much in blueness in connection with the temperature and states of excitement, but never now presented anything approaching a cyanotic condition.

I have now to relate the very extraordinary fact which is possibly explanatory of the singular condition of things just described. It will have been noticed that the chief defects present in the child were, an atrophic condition of the appendages of the skin and its accessory cellular tissue and fat, which became especially conspicuous in the absence of the scalp hair. With this we had a well-developed condition of the male sexual organs and an absence of the mammary glands and nipples. Now the mother of this child from the age of six to the present time had worn a wig on account of alopecia areata. At the age mentioned she began to lose her hair, which had previously been plentiful, in patches. She described the usual course of things, how the patches increased, and the whole scalp became smooth and bald, and how subsequently the eyebrows and eyelashes fell. After a considerable time her eyebrows and eyelashes grew again, and a few tufts of hair appeared on the scalp. But she had never regained her scalp hair sufficiently to dispense with her wig, and her eyebrows were still so poor that she was obliged to colour them. Excepting this alopecia she had no signs of deranged nutrition, being a florid, comely, well-developed

woman. The little boy was her first and only male child, but he had five sisters, all older than himself and all of whom had excellent development of hair.

Very curious speculations suggest themselves in connection with Darwin's theory of pangenesis. Under this hypothesis it may perhaps be possible that the germinal elements of the child's cutaneous system, and especially for his scalp, were derived from his mother, and were, in connection with her long baldness, very defective in vigour. With this would fit the entire absence of the mammary glands and their nipples; with this also would fit the normal development of the male genital organs and their skin, since he would be supposed to take these from his male parent. The fact that all his sisters had good development of scalp hair may be supposed to be explained by the suggestion that they inherited chiefly from their father.

It is to be added that the marriage was not one of consanguinity, and that no baldness or defects of development had been known in the family previously.

I may have perhaps a little over-stated the general absence of subcutaneous fat. Excepting on the head and hands, it was nowhere quite absent; and this remark especially applies to the abdomen and back. The deeply placed fat was less affected than the superficial. Thus, lumps of it could be detected at the root of the neck. The skin was everywhere destitute of natural elasticity and plump firmness, and where not dusky had an earthy pallor. The eyelashes were present but very weak. The eyebrows almost entirely absent.

I was indebted to Dr. Jago, of Mulgrave Place, Plymouth, for the opportunity of seeing this child and for some facts as to its history.

Remarks.—I prefer, for the present at least, to leave the above remarkable case without attempting to contrast it with other examples of congenital alopecia on record. From all these it differs, so far as I am aware, in the fact that the female sex organs (the mammæ) were absent, whilst the skin of the male sex organs was the only part

of the integument in a normal condition. These peculiarities become of the greatest possible interest when we remember that he appeared to inherit his defect from his mother. I am well aware that the explanation hinted at is a mere conjecture, and that there are a multitude of facts which might seem to militate against it. We cannot afford, however, in investigating the very difficult subject of hereditary transmission, to neglect any hint which the facts of pathology may offer. I need scarcely say anything as to the well-known law that defects, the result of disease or injury occurring in the parent and not congenital, are not transmitted to offspring. Everyone knows that circumcised fathers beget children in whom the prepuce shows no modification. To this law the case I have recorded seems to offer an exception, for there was not the slightest doubt that the mother's loss of hair was caused by the common form of alopecia areata, and did not begin till she was six years old. In fact, her hair grew again several times after its first falling, and again came off. Some will probably be inclined to consider that the mother's condition and that of her only son were associated as a mere coincidence and that the one was in no way dependent on the other. It is indeed precisely because this connection seems so probable, whilst it is in flat contradiction to received opinions, that I have thought the case worthy the attention of the Society.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' New Series, vol. ii, p. 116.)

THE

MORBID ANATOMY AND PATHOLOGY

OF

ENCYSTED AND INFANTILE HERNIA.[1]

BY

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Received January 22nd—Read May 25th, 1886.

CONSIDERABLE surgical importance may be claimed for anything which pertains to the subject of hernia. Hardly any affection is so common or more frequently demands surgical interference, and the simplest case may, when operated upon, present the most disconcerting peculiarities. It might be urged that some of these are so rare as not to be of practical importance, but if such an argument as this possesses any weight it would not apply to encysted hernia.

Every writer upon general surgery describes this variety of the disease, and testifies how interesting it is to all who are engaged in the practical duties of their profession.

The history of this affection is by no means difficult

[1] The terms "encysted" and "infantile" are in the following pages considered to indicate a purely anatomical condition.

to trace, for unlike that of congenital hernia it has never been the subject of any dispute. Sir William Lawrence says[1] that it was first described by Hey, who met with an example of it in 1764. Sir Astley Cooper,[2] in his magnificent work, alludes to Hey's observations and depicts what may be considered to be a typical specimen.

Writing in 1838 Sir William Lawrence does not allude to any other observations except these, and the knowledge of this author was so profound that it may be assumed that none other existed. Chelius,[3] and it may be said South,[3] writing in 1847, merely refer to the authors which have been mentioned, and make no addition to the subject, and the same may be said of Meckel,[4] whom they quote. Since that time, although a diligent search has been made, I am unable to ascertain that any fresh knowledge has been gained.[5] Recent authors may have made here and there new statements, but not such as will bear strict investigation. Indeed, a critical examination of the most authoritative accounts of the anatomy and pathology of encysted hernia reveals many discrepancies and leaves much to be explained. In order to justify this assertion, and because their statements are often misrepresented, it may be best to note, as briefly as is consistent with exactitude, the views which the most eminent writers have formulated, and at the same time an adequate idea may be formed as to what is usually meant by the term "encysted hernia." Fortunately this, so far as authorities are concerned, is not a very formidable undertaking. It may be deemed sufficient if I mention what Hey and

[1] Lawrence, Sir William, 'A Treatise on Ruptures,' 5th ed., 1838, sec. 2, p. 576.

[2] Cooper, Sir Astley, 'The Anatomy and Surgical Treatment of Abdominal Hernia,' 2nd ed., 1827, p. 74, pl. xi, fig. 1.

[3] 'Chelius' System of Surgery,' South, vol. ii, p, 59, 1847.

[4] Meckel, 'Handbuch der pathologischen Anatomie,' vol. xi, pl. 1, pp. 379 and 380, Leipzig, 1816.

[5] This applies to the writings of Vidal, 'Traité de Pathologie Externe,' tome iv, 1861, and to those of Th. Kocher, 'Handbuch der Kinderkrankheiten,' Tubingen, 1880, "Articles on Hernia," i, 747, *et seq.*

Cooper have said and then refer to more recent writers. The case which Mr. Hey[1] met with, and to which he gave the name "infantile hernia," was that of a child fifteen months old, and after remarking that the cæcum and beginning of the ilium were contained in the hernia, this author proceeds to say, "I found that the tunica vaginalis was continued up to the abdominal ring, and inclosed the hernial sac; adhering to that sac, by a loose cellular substance, from the ring to within half of an inch of its inferior extremity. . . . . The interior or true hernial sac was a production of the peritoneum as usual, and contained only the cæcum or head of the colon. . . . Having removed the proper hernial sac I examined the posterior part of the exterior sac; and found it connected with the spermatic vessels in the same manner as the tunica vaginalis is, when the testis has descended into the scrotum." Everything that this eminent surgeon says about the tunica vaginalis in this description is quite clear and precise, but as regards the true hernial sac his remarks are, so far perhaps, slightly wanting in precision. For instance, it is not said whether its walls were constructed of one or more than one layers of peritoneum, and yet it will be seen presently that this is a most important question. However, Hey explains the pathology of the disease in the following way:[2] "In the fœtus a process of the peritoneum is brought down, through the ring of the external oblique muscle of the abdomen, by the testicle as it descends into the scrotum; which process forms an oblong bag communicating with the cavity of the abdomen, by an aperture in its upper part. This aperture is entirely closed at, or soon after, birth. The upper part of the bag then gradually contracts itself, till the communication between that portion of it which includes the superior and greater part of the spermatic chord, and the lower part of the bag, which includes the testicle and a small share of the chord, is obliterated. The lower part of the process

[1] Hey, 'Practical Observations on Surgery,' 3rd ed., 1814. An account of an uncommon species of scrotal hernia, p. 226, *et seq.* [2] Ibid., pp. 228 and 229.

or bag retains its membranous appearance, and is called tunica vaginalis testis propria; while the upper part covers an irregular cellular substance, without any sensible cavity, diffused amongst the spermatic vessels, and connecting them together.

"In the hernia which I am describing, the intestine was protruded after the aperture in the abdomen was closed; and therefore the peritoneum was carried down along with the intestine, and formed the hernial sac. It is evident, also, that the hernia must have been produced while the original tunica vaginalis remained in the form of a bag as high as the abdominal ring: on which account that tunic would receive the hernial sac with its included intestine; and permit the sac to come into contact with the testicle. The proper hernial sac, remaining constantly in its prolapsed state, contracted an adhesion to the original process of the peritoneum which surrounded it, except at its inferior extremity: there the external surface of the hernial sac was smooth and shining, as the interior surface of the tunica vaginalis is in its natural state."

Before making any comments upon this very clear statement perhaps it will be best to recount the views of another writer whose name has been prominently associated with this subject.[1]

Sir Astley Cooper,[2] describing what he terms an encysted hernia, says: "On opening the tunica vaginalis, instead of the intestine being found lying in contact with the testicle, a second bag or sac is seen inclosed in the tunica vaginalis, and enveloping the intestine. This bag is attached to the orifice of the tunica vaginalis, and descends from thence into its cavity; it generally contracts a few adhesions to the tunica vaginalis, while its interior bears the character of a common hernial sac.

[1] Mr. Birkett, article on "Hernia" in 'Holmes's System,' 3rd ed., vol. ii, 1883, p. 807, &c., says that "Infantile hernia of Hey and encysted hernia of the tunica vaginalis of Astley Cooper are synonymous terms" (see also Mr. Wood's remarks at p. 485).

[2] Cooper, 'Anatomy and Surgical Treatment of Abdominal Hernia,' pt. 1, 2nd edit., 1827, p. 79.

"The idea which I have formed of the nature of this variety of hernia is, that the tunica vaginalis, after the descent of the testis, becomes closed opposite the abdominal ring, but remains open above and below it. The intestine descends into the upper part, and elongates both the adhesion and tunica vaginalis, so as to form it into a bag, which descending into the tunica vaginalis below the adhesion, and becoming narrow at its neck, though wide at its fundus, receives a portion of the intestine, which in the following case was too large either to be returned into the abdomen, or to retain its functions whilst it continued in the sac."

The cases which Sir Astley quotes were met with by his colleagues in patients upon whom they operated, but only one of these was verified by a post-mortem examination. It will not be necessary to repeat Forster's description, which Sir Astley quotes. He concluded it by saying that after he had opened the tunica vaginalis, and turned back its edges, there was[1] "exposed a hernial sac pendent from the ring, and descending towards the testicle." In addition Sir Astley Cooper remarks that two other encysted herniæ were met with at Guy's about that time, one during an operation, the other during dissection. It seems by no means improbable that the latter is the actual specimen which he described and depicted in his great work, and which is still to be found in the museum of Guy's Hospital.[2]

If we compare what Hey and Cooper said, it will be allowed that their views are not dissimilar. They both agree in stating that the tunica vaginalis, in the case of encysted hernia, becomes closed at its upper part, and they both attribute the formation of the hernial sac to intestinal protrusion, but neither of them makes an explicit statement concerning the composition of the hernial sac, whether it consisted of one layer of serous membrane, or

[1] Ibid., p. 80.

[2] Sir William Lawrence says that such a one was placed in the museum by Sir Astley Cooper (see Plate xi, fig. 1, Cooper on "Hernia").

of more than one. There is, however, one very important circumstance to which I would draw attention. Hey, in describing the closure of the tunica vaginalis, says nothing whatever about adhesions, but simply states that "it gradually contracts itself." Cooper, it will be remembered, says, "The tunica vaginalis, after the descent of the testis, becomes closed opposite the abdominal ring, but remains open above and below." Nothing so far has been said about adhesions, but in the next sentence he remarks, "The intestine descends into the upper part (i. e. of the tunica vaginalis), and elongates both the adhesion and tunica vaginalis, so as to form it into a bag, which, descending into the tunica vaginalis below the adhesion, &c." Although this account may not be free from ambiguity, yet it implies that adhesions closed the tunica vaginalis, and that they actually entered into the formation of the hernial sac. Without assuming that this interpretation of Sir Astley Cooper's statement is correct, I will proceed to quote what has been written by authorities who have succeeded him, but before doing so it is significant to observe that Sir William Lawrence neither refers to, nor passes any opinion upon, Sir Astley Cooper's statement. Mr. Birkett,[1] moreover, simply says that the ventral orifice of the processus vaginalis becomes closed, "but the canal persisting from that point to the testis. The hernia slowly pushes before it the parietal peritoneum of the abdomen into this sheath, and when the parts are dissected it is seen that the tunica vaginalis is continued up to the abdominal ring, and encloses the hernial sac, as Mr. Hey describes." Mr. Birkett, it is superfluous to point out, does not mention adhesions in connection with the true hernial sac, and merely remarks incidently that it is made of serous membrane; he does not give a detailed account of its structure.

Although at the risk of wearying the reader by constant repetition, yet since it is conducive to a clear conception

[1] Birkett, 'Holmes's System of Surgery,' 3rd ed., 1883, p. 807, *et seq.*, vol. ii.

I would venture to quote more authorities upon this subject.

Writing in the present year (1885), Mr. John Wood[1] says as follows: "The canal of Nuck [processus vaginalis testis] becomes closed first at the deep ring, leaving a cicatrix which is always more or less traceable. The obliteration extends down the cord to within half an inch of the testicle. The serous membrane degenerates and is transformed into connective tissue, which more firmly binds together the elements of the cord. Sometimes the obliteration extends only to the parts near the deep ring. Then, while the cicatrix is still weak, some violent crying or coughing efforts of the child protrude the bowel, pushing and dilating the cicatrix before it, and a fresh sac of peritoneum is invaginated from above into the upper part of the large tunica vaginalis, which is pushed before it into the scrotum. We have thus formed that kind of children's rupture with a double sac which is called infantile [or encysted] hernia, fig. 1130 (*v.* Fig. 1, p. 486). In this there are three layers of serous membrane placed in front of the bowel in the scrotum, viz. two layers of the invaginated tunica vaginalis, and one of the fresh, or real sac of the hernia.

The expression "pushing and dilating the cicatrix before it," which Mr. Wood uses in describing the way in which the extruded bowel forms the hernial sac, certainly leaves an impression upon the mind that the hernial sac may be formed, in part at least, of cicatricial tissue. The very clear figure (Fig. 1, p. 486) which accompanies the description would seem to show that the sac which contained the hernia consists of two layers of serous membrane, and that the original communications between the tunica vaginalis and the peritoneal cavity had become entirely obliterated, in truth, it answers perfectly to the graphic description of Forster[2] "A hernial sac

[1] Article on "Hernia," Ashhurst's 'Encyclopædia,' vol. v, p. 1132, fig. 1130, 1885. I am indebted to Mr. Wood for permission to reproduce this diagram (*v.* Fig. 1, p. 486).

[2] Cooper, loc. cit., p. 79, *et seq.*

Fig. 1.

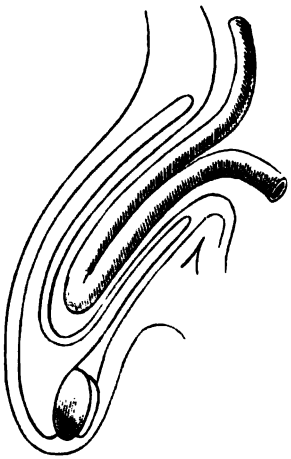


Diagram of infantile (or encysted) hernia. Copied from fig. 1130. Ashhurst, vol. v (Wood).

pendent from the ring, and descending towards the testicle."

It can hardly be denied that a perusal of these various quotations leaves an impression that the authors of them seem to imply that, in some way or other, cicatricial tissue enters into the composition of the sac of an encysted hernia, but should any doubt remain upon this point it may be dissipated by referring to the writings of Mr. Timothy Holmes. Speaking of this variety of hernia this author says,[1] "This may occur in consequence of adhesions having obstructed the neck of the infundibuliform process and formed a membrane. This membrane being distended by the protruding bowel, forms a hernial sac for it."

Leaving aside for a moment the question of the cicatrix, it cannot be doubted that Mr. Wood and Mr. Holmes describe and delineate that which most surgeons would

[1] 'A Treatise on Surgery,' T. Holmes, 1882, p. 647, fig. 312. The diagrams which Mr. Holmes gives are, by his kind permission, introduced in Figs. 2 and 3, pp. 488 and 492.

consider a representative encysted hernia.[1] However this may be, Mr. Erichsen depicts and describes quite a different variety. Since Mr. Erichsen's account is a very brief one perhaps it may be given. "Encysted hernia of the tunica vaginalis, or infantile hernia, as it has been somewhat absurdly termed, occurs in those cases in which the funicular portion of the tunica vaginalis is partly obstructed by a septum, or by being converted into filamentous tissue, but in such a way as to leave a pouch above, which is protruded down behind or into the tunica vaginalis, so that it lies behind the cavity."

The last sentence certainly admits two alternatives; in one event the hernial pouch may bulge into the tunica vaginalis, and in the other simply lie behind it; the latter is probably the case which he depicts.[2] But, although the words "septum" and "filamentous tissue" are met with in this account, used in connection with the method of closure of the funicular portion of the tunica vaginalis, yet it is not clearly stated what those structures may have to do with the formation of the hernial sac. In any case, judging from the diagram, we have now to deal with a hernia quite different from that which, up to this point, has been referred to, unless it be thought that Mr. Erichsen's account tallies with that which has been quoted from Hey.

A glance at Mr. Erichsen's diagram shows how much it differs from that which has been taken from Mr. Wood's writings. It would not appear necessary to attempt to reconcile these conflicting authorities, for, according to Mr. Timothy Holmes,[3] they are both correct. This author figures and describes two varieties of encysted hernia; one, already mentioned, like Mr. Wood's, a hernial sac pendant from the ring, the other, like Mr. Erichsen's, a pouch behind the open processus vaginalis.

[1] See also Bryant, 'The Practice of Surgery,' ed. iv, vol. i, 1884, p. 732, fig. 264.

[2] Erichsen, 'The Science and Art of Surgery,' vol. ii, ed. 8, p. 816, fig. 797.

[3] Loc. cit., p. 647, figs. 311 and 312 (for copies, see Figs. 2 and 3, pp. 488 and 492).

FIG. 2.

Diagram (copied from Holmes) whose description is as follows:—"Another variety of infantile hernia (the encysted form). The bowel instead of passing behind the closed funicular process has distended the membrane which closes its upper end, and has pushed itself into the funicular process, the upper or back wall of which envelopes it. In this case, therefore, the hernial sac is furnished by the funicular process itself, and only two layers of peritoneum cover the intestine."

There can be little doubt but that Mr. Holmes has expressed the usually accepted views upon this point; and most surgeons and pathologists would concede that there are, in fact, two varieties of encysted hernia. Mr. E. Owen,[1] who met with an example of the disease, which will be mentioned presently, is of this opinion, and his book upon children's diseases affords very clear diagrams of the two varieties. In order to avoid confusion it will be best to mark each of these varieties of encysted hernia with a definite name. Those which Mr. Holmes uses, although perhaps open to objection, will serve the purpose.[2] In the first place the term "encysted hernia" will be applied to the condition in which, when the unobliterated processus vaginalis is opened,[3] a hernial sac is seen pendant from the internal ring; and secondly, the term "infantile hernia" will be applied to those cases in which, when the unobliterated processus vaginalis has

[1] 'The Surgical Diseases of Children,' 1885, p. 345, figs. 57-8.

[2] Holmes, p. 647, figs. 311 and 312.

[3] The term "processus vaginalis" is applied to the process of peritoneum which accompanies the transition of the testis, and which afterwards becomes tunica vaginalis propria and ruins processus vaginalis.

been opened, a hernial sac or pouch is found behind it, and bulging into it.

Without endeavouring at present to determine to which of these varieties the herniæ described by Hey and Cooper belonged, it may be remarked that most of the authorities who have been mentioned confine themselves, so far as I can judge, to the elucidation of the anatomy and pathology of the encysted form. With regard to the other sort, the infantile, it is true that Mr. Erichsen figures it, but Mr. Holmes throws a certain doubt upon its genuineness, for he says that the diagram which he gives is intended to represent "the *assumed* condition of the parts in infantile hernia." However, he proceeds to discuss the manner of its formation, and says, "In this form the communication between the peritoneal cavity and the infundibuliform process leading into the tunica vaginalis is obstructed at or about the external (or superficial) ring, but the process itself is not obliterated, so that the cavity of the tunica vaginalis extends up to the external ring. Then a hernia comes down and generally slips behind this upper prolongation of the tunica vaginalis (fig. 311)."[1] (See fig. 3, p. 492.)

This completes a summary of the current views upon the subject of encysted and infantile hernia. With all due deference one cannot help saying that when they are submitted to a critical examination they will be found wanting in scientific precision. In order to support this opinion I will confine myself, for the present, to the pathology of encysted hernia, and without further preliminaries, discuss a question which seems to go to the very root of the matter; and it is this: What has cicatricial tissue to do with the formation of the hernial sac? It cannot be denied that although Hey said nothing whatever about cicatrices, adhesions, or septa, yet we find them mentioned by Sir Astley Cooper and succeeding authors, until at last the greatest importance seems to be attached to them.

[1] Ibid., p. 648, fig. 311.

In order to determine this most important question two methods of investigation are open to us: in the first place, to inquire whether the upper part of the processus vaginalis is ever closed by adhesions or cicatrices capable of forming a septum suitable for the creation of a hernial sac; and next, to see whether the sac has the appearances which it might be expected to possess had it been formed of cicatricial tissue.

With regard to the first part of this inquiry, it seems very hard to discover upon what exact basis of fact the actual existence of the septum, which is assumed to close the processus vaginalis, rests.

I have been unable to discover that any author says that he has actually seen such a thing. Although it is a hopeless task to try and prove a negative, yet it cannot be without influence upon this argument to notice that Wrisberg,[1] Seiler and others investigated the processus vaginalis with great industry, and that none of them mention such a thing, and it is hardly in accordance with our general knowledge of tubes with endothelial linings to conceive of their closure by septa. The function of the processus vaginalis is to give passage to the testicle, and when it has done this it not only ceases to grow, but undergoes retrograde atrophic changes. Under the circumstances we are considering, in which it becomes the receptacle for an encysted hernia, the very opposite occurs; the processus vaginalis grows and its lumen increases, a fact which diminishes the likelihood of its occlusion by a septum. It must be confessed that an inspection of Cloquet's drawings[2] suggests very strongly, whatever normal anatomy may afford or *a priori* reasoning suggest, that, nevertheless, hernial sacs may be partitioned by septa. From a septum in a hernial sac to one in the processus vaginalis is not a

[1] "De testiculorum ex abdomine in scrotum descensu, etc.," 'Comment. Soc. Reg. Scient. Gotting.,' 1800, p. 178, *et seq.* 103 examinations are recorded by Wrisberg, and Mr. Birkett attributes 54 to Camper and 21 to Seiler (art. in 'Holmes's System,' 3rd edit., vol. ii).

[2] 'Recherches sur les causes et l'anatomie des hernies abdominales,' Paris, 1819.

long leap. However, an examination of Cloquet's specimens themselves, which are in the Dupuytren Museum,[1] shows that any partial septa which are present in them are really due to pleats in the walls of the hernial sac, each accompanied by a corresponding constriction upon the exterior, and very like the folds of the large intestines.[2] It would not be right to draw definite conclusions from these specimens, because they are simply hernial sacs which have been dried and varnished, but they hardly suggest the existence of septa of cicatricial tissue. It is true that they show complete constriction of the hernial sac;[3] but even in this case, I do not think it has ever been argued seriously that an encysted hernia could be produced by an intussusception of one part into another, for, owing to the gradual nature of the constrictions, this would seem an impossibility.

It is not for a moment pretended that the arguments which have just been adduced, prove the impossibility of the processus vaginalis ever being occluded by a cicatricial septum, but it can hardly be denied that they suggest the improbability of such an event. However this may be, under these circumstances it seemed best that they should be stated, for the sequel will show that the pathology of encysted hernia depends more upon the whole weight of evidence than upon any particular fact. The reason for this will be clear when we begin to array the evidence which has been afforded by an examination of the various specimens of encysted hernia which are to be found in the various London museums, and owing to the very great kindness and courtesy of the curators, I have been permitted to dissect and examine them at my leisure. In order to avoid the embarrassment which the multiplication of intricate details sometimes causes perhaps I may be permitted to begin with a general statement of results.

Just for the moment it may be said that the various

[1] Cloquet's specimens are Nos. 269 to 315.

[2] See Specimens 236 and 306.

[3] See Specimens 310, 312, 314, and 315.

specimens seem to belong to two very distinct types. In both of these it is an essential feature that the sac of the tunica vaginalis should be very large, reaching almost, if not quite, as far as the peritoneum ; but the question of its communicating with the cavity of that membrane is a point which will be discussed presently. This much having been premised, it may be stated that the two apparent varieties are those which have been already spoken of as "encysted

FIG. 3.

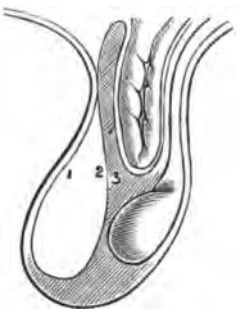


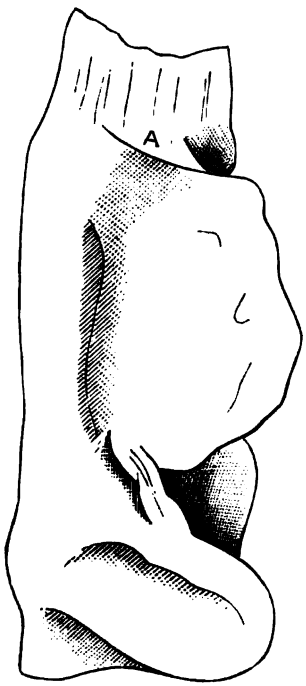
Diagram (copied from Holmes, fig. 311) of the (assumed) condition of the parts in an infantile hernia. The tunica vaginalis (1) is closed above, at or near the external inguinal ring, but its funicular portion is open. The bowel in the hernial sac lies behind this funicular portion, and is represented in the diagram as having made its way between the funicular process and the cord. The relation of the sac to the cord seems, however, to be variable. The bowel is covered in cutting down from the skin by three layers of peritoneum, viz. 1 and 2, the opposite surfaces of the funicular process, and 3 the anterior layer of the peritoneal hernial sac.

hernia" and "infantile hernia." Specimens 2497 and 2947[50] in the Guy's Hospital Museum (*v.* Figs. 5 and 6, pp. 495 and 498) and Specimen C. D. 20 in the Museum of St. Mary's Hospital (*v.* Fig. 8, p. 510) may be considered representative of the encysted. Indeed, there is not much doubt but that one of them, Specimen 2497[50], Guy's, is the very one which Sir Astley Cooper depicted ;[1] whilst the St. Mary's specimen was described as an encysted hernia

[1] Cooper on "Hernia," plate xi, fig. 1.

in the British Medical Journal.[1] The second variety of encysted hernia which the museums contain is clearly of the sort which has already been described under the name

FIG. 4.[2]



Drawing of an infantile hernia, specimen R. 24, in the St. Thomas's Hospital Museum. The bulging of the hernial sac into the tunica vaginalis is shown and also the fold (plica vascularis), which extends from lower extremity of the sac to the epididymis. In the catalogue the specimen is named, "encysted." A, mouth of sac.

of infantile, and which consists of a pouch or bag of peritoneum pushed down behind the greatly enlarged tunica vaginalis.[3] Specimens 2488[50] in the Guy's Hospital

[1] 'British Medical Journal,' Aug. 1, 1874, p. 140, E. Owen.

[2] I am indebted to the kindness of Mr. Shattock for permission to examine and draw this specimen.

[3] As Hey describes.

Museum and Specimen R. 24 in the Museum of St. Thomas's Hospital (*v.* Fig. 4, p. 493) may be considered representative of this class.

It may be remembered that the current views as to the pathology of these two sorts of encysted hernia have already been stated. With regard to the first, it has been shown that there is a strong impression that cicatricial tissue enters largely, if not entirely, into the formation of its sac. Two arguments have already been advanced to show the unlikelihood of this being true; first, the *a priori* improbability of a growing processus vaginalis, the lumen of which has been enlarged, ever being occluded by a septum of cicatricial tissue, suitable for becoming a hernial sac; secondly, the fact that such a septum has never actually been seen. Now, since it can hardly be denied that the specimens which have been chosen are typical, we may proceed to inquire whether they confirm or contradict the preceding propositions. The two specimens in the Guy's Hospital Museum[1] show no indication that cicatricial tissue has entered into the construction of the hernial sac, and the same may be said of the St. Mary's specimen,[2] which will be referred to afterwards at length. So far as I can ascertain two distinct layers of serous membrane form the sac walls of these encysted herniæ. Of these two layers, that which lines the interior of the sac is continuous with the peritoneal cavity, whilst that which covers its exterior is continuous with, and forms part of, the tunica vaginalis.[3]

The real importance of this observation will be clearer after awhile, but for the moment we may pause to meet an argument which readily suggests itself, namely, whether after a time even a septum of cicatricial tissue might not assume the characters of the serous membranes in its neighbourhood.

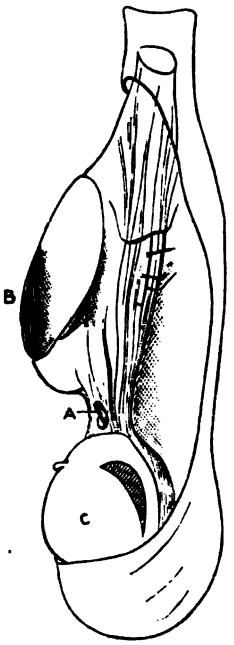
[1] Figs. 5 and 6, pp. 495 and 498.

[2] Fig. 8, p. 510

[3] Approximating very closely the condition described by Hey, v. a. pp. 481 and 482.

There is no proof that such an event takes place under any circumstances, and an examination of the specimens (Figs. 5, 6 and 8) affords no evidence in its support. Not only are the two layers of serous membrane of which the

FIG. 5.[1]



Description of Fig. 5 of "encysted hernia" (v. Catalogue), No. 2497, Guy's Hospital Museum. The two layers which form the sac wall are shown and also the band which passes from the epididymis to its extremity. In the interior of the sac of this hernia, behind, there is a curious pouch made by a transverse fold of serous membrane. B, hernial sac. A, band with spermatic artery in its midst. C, testicle and epididymis.

true hernial sac is composed quite distinct, but there is muscular tissue between them; a point which will be explained later.

[1] Dr. Goodhart kindly permitted me to examine and draw this and other specimens.

If it is clear that the sacs of these *encysted* herniæ consist not of cicatricial tissue but of a double layer of serous membrane we may now proceed to investigate the crucial question, whether the tunica vaginalis in these cases of encysted hernia communicates with, or has been shut off from, the peritoneal cavity. Allowances must be made, in investigating this, for alterations produced by previous dissection or by operations. The possibility of adhesions having been destroyed by this means is too obvious to need pointing out. Without doubt the front of the upper edge of the hernial sac in the St. Mary's specimen (*v.* Fig. 8, p. 510) was closely applied, perhaps adherent, to the wall of the tunica vaginalis, but I am of opinion that in it the processus vaginalis communicated with the peritoneal cavity by a wide opening, and I think the same statement may be made with regard to another of these encysted herniæ (No. 2497, Guy's), see fig. 5. In a specimen which more than any other might be called a "hernial sac pendant from the ring" (*v.* Fig. 6, p. 498, Sp. 2497[50], Guy's), the tunica vaginalis is open right up to the neck of the sac, but at that point its walls adhere to one another. This adhesion is so slight and the continuity of the serous membrane is so palpable, that if the smallest pressure were made with a probe the attachments would be loosened, and the specimen as regards the relations of the neck of the sac made like an infantile hernia (*v.* Figs. 3 and 4, pp. 492 and 493).

Having now ascertained the condition of the tunica vaginalis in the most typical encysted herniæ, it is unnecessary to say that the opinions which have been quoted concerning the pathology of this disease are unacceptable.

If the various specimens of encysted hernia were diagrammatically represented, it would be seen that they belonged to the infantile type (Fig. 3).

In either case the hernial sac consists of an outer and an inner layer of serous membrane, one formed by a protrusion from the peritoneum, the other by the tunica vaginalis. The differences which are present depend upon the degree

to which the hernial sac may have bulged into the tunica vaginalis and not to any difference in their actual construction.

Of course this takes for granted that the existence of the infantile variety is admitted and its morbid anatomy acknowledged, but, upon this point, an inspection of the specimens in the various museums leaves absolutely no doubt, and the facts which have been mentioned tend to justify this assertion. The truth of the statement that all the specimens of encysted hernia belong to the sort called infantile, would not be at all obvious if it depended upon a comparison instituted between what may be called exaggerated instances: for example, if an infantile hernia which hardly bulges at all into the tunica vaginalis be compared with one which protrudes excessively (*e. g.* compare Figs. 4 and 6).

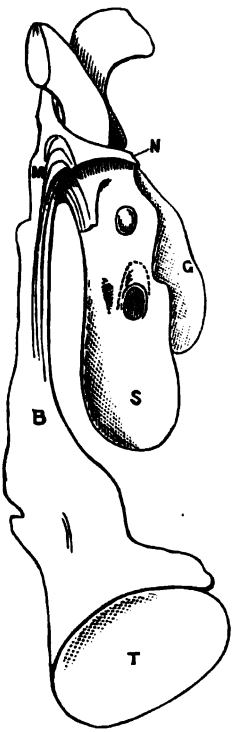
But between these extremes intermediate grades exist, and from these a series may be constructed to illustrate the progression from one to the other. Perhaps it is unnecessary at present to do more than mention a specimen of infantile hernia (Sp. R. 24, St. Thomas's Hosp., Fig. 4) which, although typically belonging to the infantile variety, has many of the characters attributed to the so-called encysted.

Before concluding this account of the morbid anatomy of the encysted herniæ, their relation to the posterior wall of the tunica vaginalis may be mentioned. It has been stated that the degree in which the hernial sac protrudes into the tunica vaginalis varies in different specimens, and so far, perhaps, as concerns those which bulge least, nothing requires to be said. However, when the protrusion is considerable, the cyst-like sac is attached to the posterior wall of the tunica vaginalis by a mesentery which extends along the whole length of its posterior surface. This may have been so in Hey's case, although he assumes that the attachment was merely an adhesion formed after the occurrence of the hernia (vide p. 482). It may be added that it is usual to find that the lowest part of this

mesentery attaches the hernial sac to the epididymis, forming a fold (plica vascularis) the importance of which will be explained.

Only one specimen seems to contradict this assertion

FIG. 6.



"Encysted Hernia" (*v.* Catalogue), No. 2497[50], Guy's Hospital Museum. Showing attachment of sac to the posterior wall of the processus vaginalis; also muscular fibres turning round fornix between the sac and vaginal process. A curious little pouch is seen upon the wall of the hernial sac.

M, muscle-fibres; N, neck of sac; G, contents, gut; B, cord; S, hernial sac; T, testis. (This is probably the specimen delineated by Sir Astley Cooper, Plate xi, fig. 1.)

and it is depicted in Fig. 6, but the difference is more apparent than real, and is due to the extraordinary way in

which the sac has been protruded into the tunica vaginalis.

That this view is correct will, I think, be clearly shown when the pathology of this affection is discussed.

Having endeavoured to describe the morbid anatomy of the most typical examples of encysted hernia, and having sought to show that they belong to the infantile variety, perhaps it may be as well before advancing any facts concerning their pathology to recapitulate the arguments which have been used to contradict the usual opinions upon the subject.

1. The absence of proof that the processus vaginalis is ever closed by a septum of cicatricial tissue.

2. The improbability of a septum being formed in a processus vaginalis which has presumably grown, and the lumen of which has increased.

3. That the sac of an encysted hernia does not consist of cicatricial tissue, but of two layers of serous membrane.

4. That it is doubtful whether the processus vaginalis is invariably shut off from the peritoneal cavity in these cases, or if it be shut off, the closure is effected in such a way as to exclude the possibility of a septum of cicatricial tissue having existed.

To these destructive arguments may be added the constructive ones which are contained in the descriptions of the various specimens, and as we proceed to discuss their pathology others will be forthcoming.

The various authors who have written upon the pathology of these herniæ have confined their remarks to speculating on the causation of the encysted variety. As far as I am able to judge, the tendency has been to attribute the latter to modifications which take place in cicatricial tissue which is supposed to obstruct the processus vaginalis. Assuming that this "theory" has, in the preceding pages, been disproved, and that it has been substantiated that all the specimens belong, in reality, to the infantile variety, we may now proceed to inquire how infantile hernia is produced. With the exception of Mr. Hey's observations,

already quoted, and which probably apply to this condition, authorities say but little. Mr. Holmes says that their origin is a hernia which slips behind the upper prolongation of the tunica vaginalis. This is hardly an explanation of the pathology of infantile hernia, and, in the absence of any other, nothing remains but to consult the various specimens for information as to their elucidation. Whatever help clinical history may afford in other cases, in this it is valueless. It is true that Mr. Hey's and Mr. Owen's cases happened in infants, but, as Mr. Birkett points out, infantile hernia may seem to originate for the first time during adult life. Many facts, more particularly the state of the tunica vaginalis, irresistibly suggest that infantile hernia is due to some peculiarity in the process of development. It has been remarked already that it is an essential feature in this disease that the cavity of the tunica vaginalis be of large size and either in communication with the abdomen or separated from it by the apposition and adhesion of its walls opposite the neck of the hernial sac.

Those who are acquainted with hernia into the tunica vaginalis (congenital hernia) will at once perceive that this is a condition with which they are familiar. Without doubt in cases of hernia into the tunica vaginalis the patency of that membrane is the predisposing cause of the rupture, and it must be exceedingly rare, as Kocher points out, for a protrusion to occur early enough to prevent the closure of this funicular process.

However this may be, well authenticated cases of hernia into the tunica vaginalis show that the congenital defect of patency existed long before the rupture, so that, even if it be clearly substantiated that in the case of an infantile hernia, the rupture had not shown itself until adult life, it would not invalidate the assumption that its predisposing cause was a developmental defect. An examination of the specimens of infantile hernia (including the encysted in this term) creates a very strong impression that events connected with the transition of the testicle

have a predominating influence upon the origin of the disease.

It seems reasonable, therefore, to begin with a review of the various events which are associated with that act, and afterwards inquire whether they throw any light upon this subject.

Few questions have been studied with so much care and diligence as the transition of the testis, and the result has been set forth in a formidable literature. It seems unnecessary in this place to endeavour to reconcile the conflicting statements of various authorities; they have been excellently summarised in the elaborate monograph of Godard.[1]

For the purpose of this inquiry, Mr. Curling's account[2] of the transition of the testis may be taken as a basis, for it is most in accordance with that which can be seen. As far as it seems possible to investigate this subject by dissection Mr. Curling has succeeded, and unless new methods had been adopted, little would remain to be added to his description. It is not proposed to enter into an elaborate and detailed account of the results which have been obtained by the examination of more than twenty human fœtuses of various sizes. It has been implied that, so far as concerns dissection, they confirm nearly all that Mr. Curling has said. In addition, the question has been studied in the following way, whole fœtuses were placed in a large quantity of a solution of chromic and hydrochloric acid until the soft tissues were hardened and the bones were decalcified. The whole pelvis was then suitably embedded in paraffin, and a series of thin sections cut with a large microtome.

Having mentioned these particulars, we may now proceed to sketch the result of the various investigations, but, since it is proposed to discuss this subject at greater

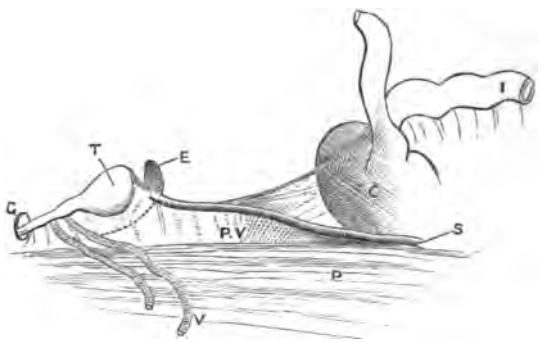
[1] Godard, "La Monorchidie et la Cryptorchidie chez l'homme," 'Comptes Rendus,' 1856, p. 315.

[2] 'A Practical Treatise on the Diseases of the Testicle,' T. B. Curling, 4th ed., 1878, p. 17, *et seq.*

length at another time, the narrative will be kept as free as possible from controversy. It will be sufficient for present purposes if the position and attachments of the testicles, as they are usually found at the seventh month of intra-uterine life, be first described.

At this time, as Fig. 7 shows, the testis is situated

FIG. 7.



Drawing made from a seven or eight months fœtus to show the fold (plica vascularis) which connects the testis with the cæcum.

T, testicles; E, epididymis; P, psoas; V, vas deferens; G, plica gubernatrix, disappearing into processus vaginalis; P.V, plica vascularis; C, cæcum; S, spermatic artery; I, ilium.

in the iliac fossa, a little above the internal abdominal ring, and is attached to the front of the psoas muscle by the mesorchium, which is simply a fold of peritoneum about one third of an inch wide. In its free border the body of the testicle and epididymis lie a little way apart, the latter being nearer the attachment. In addition, the mesorchium has two folds which extend upwards and downwards from the testicle. The upper contains the spermatic vessels and a quantity of unstriped muscle-fibres,[1] and may be called the "plica vascularis."

[1] All statements made in this paper concerning muscular fibres have been repeatedly verified by microscopic examination.

The muscle[1] belongs to the gubernaculum testis, and will be fully described hereafter. The upper part of the plica vascularis of the right side, as Wrisberg[2] states, ends either upon the vermiform appendix, the mesentery, the cæcum, or the ileum. Without doubt the main portion passes to the common mesentery, which, at this period, belongs to the cæcum and ileum, the remainder being subsidiary; on the left side the plica vascularis passes to the sigmoid flexure. The inferior fold of the mesorchium is called the plica gubernatrix, because it contains the testicular end of the gubernaculum testis. In an eight months' fœtus the lower end of the plica gubernatrix disappears into the orifice of the processus vaginalis, which has commenced to be formed. The way in which a sort of test-tube of peritoneum accompanies the transition of the testicle is too well known to call for comment, but the manner of its production requires to be described. It seems natural to suppose that the serous membrane accompanies the gland on account of their mutual adhesion. Although this may be an element in the case, another factor must be taken into consideration, for there can be little doubt that the processus vaginalis moves towards the scrotum in advance of the testicle.[3] As a rule, the peritoneal test-tube does not precede its contents by many lines, but the distance may be so palpable as to preclude the possibility of the testicle having pushed or dragged its serous covering towards the scrotum. A certain degree of support is afforded to these observations by the well-known fact[4] that when the testis is undescended a process of peritoneum may reach towards the scrotum. This is shown in many museum specimens (*e. g.* 2339^{30}, 2339^{50}, 2339^{25} in the Guy's Hospital Museum,

[1] This may be the fold sometimes named after Seiler, see Banks, 'On the Wolffian Body, &c.,' Edinburgh, 1864, but Sappey calls the whole mesorchium "Seiler's fold," 'Traité d'Anatomie,' vol. iv, p. 604.

[2] Loc. cit., p. 230.

[3] Quain's 'Anatomy,' 9th ed., vol. ii, p. 908.

[4] Lawrence, p. 569, also Cloquet, p. 23 ('Les Causes,' &c.).

also Sp. 91, S. IX in the St. George's Hospital Museum). Since in some of these cases the testicle is adherent in the iliac fossa, it is obvious that it could not have pushed down the peritoneum. If the superior terminations of the gubernaculum be examined, both anatomically and microscopically, the reason why the processus vaginalis moves in advance of the testicle is explicable. The fibres of that muscle are inserted, not only into the epididymis, vas deferens and testicle, but also into the peritoneum. At about the seventh month of intra-uterine life, muscular fibres may be seen inserted into the extremity of the processus vaginalis, and, moreover, many of them are prolonged up the mesorchium into the plica vascularis, and so onwards to the peritoneum which lines the posterior wall of the abdomen. The lower attachments of the gubernaculum are described so clearly by Mr. Curling that a detailed description seems unnecessary. It is generally recognised that it has three main attachments; one to the abdominal wall; another to the pubes, the lower part to the sheath of the rectus and the root of the penis; and a third to the bottom of the scrotum. Repeated dissections substantiate these statements. Perhaps it may be mentioned that some of the fibres of the portion which mingles with the wall of the abdomen pass downwards into Scarpa's triangle and are not unimportant in affording a plausible reason for the occasional passage of the testicle into the thigh.[1] It is quite unnecessary to say that the function of pulling the testicle into the scrotum is attributed to these divisions of the gubernaculum. The first pulls it as far as the internal abdominal ring, the second to the pubes, and the third deposits it in its final resting place.

If we proceed to consider the various events which accompany the transition of the testicle, I think it will be admitted that the gubernaculum must exert a certain

[1] Mr. McCarthy mentions this occurrence, but attributes it to abnormal fibres of the gubernaculum, Quain's 'Dictionary of Medicine,' 1882, p. 1606.

amount of force. For instance, if a fœtus be chosen in which the gland is about to pass through the abdominal wall, and traction be made upon the gubernaculum, it is clear that as the testicle travels towards the scrotum not only the mesorchium and its contents and the processus vaginalis, but the peritoneum which lines the posterior wall of the abdomen, moves with it. In consequence of this locomotion of the serous membrane, the cæcum and ileum on the right side, and the sigmoid flexure upon the left, attain a lower position in the abdomen, a circumstance upon which both Scarpa[1] and Wrisberg[2] have commented. That the transition of the testicle has an important influence upon the movements of the viscera is suggested by the fact that in the cases of retained testicle the cæcum may fail to complete its descent into the iliac fossa[3]. The exact contrary of this may happen, and the cæcum or the ileum be dragged with the testicle into the scrotum, producing a congenital cæcocele. Wrisberg,[4] Scarpa[5] and Cloquet[6] mention such cases and say that the cæcum was attached to the testicle by a fold which they identify as the plica vascularis, but without naming it. I have been so fortunate as to find a congenital cæcocele in a very young infant. In it the plica vascularis had entirely disappeared, but upon the back of the hernial sac there was a quantity of muscular fibres and fibro-areolar tissue, which passed from the back of the testicle upwards to the cæcum.[7] These bands were parallel to and adjoining the spermatic vessels, and without doubt the hypertrophied representatives of those of the gubernaculum which before

[1] 'A Treatise on Hernia,' translated by Wishart, Edinburgh, 1814, p. 38.

[2] Loc. cit., p. 230.

[3] See a paper by author, 'Brit. Med. Journ.,' Sept., 1882, p. 575, "Abnormalities of the Cæcum and Colon with Reference to Development."

[4] Loc. cit., p. 233.

[5] Loc. cit., p. 203.

[6] 'Causes, &c., des Hernies,' p. 23. See also Cruveilhier, 'Anatomie Pathologique,' vol. iii, p. 307, Paris, 1849.

[7] My friend Mr. D'Arcy Power kindly verified this fact.

birth normally exist in this situation. It would be illogical to argue that because these were present therefore they were responsible for the abnormal descent of the cæcum, but it is not impossible. This specimen is important in other respects and will be mentioned again. Assuming it is true that a general locomotion of the peritoneum of the back of the lower part of the abdomen accompanies the transition of the testicle, it remains to be decided whether the gubernaculum is capable of such an effort. Judging from the amount of its muscularity this question may be answered in the affirmative, but it is doubtful whether all of its attachments are adequate. It is easily appreciated that the portions which adhere to the abdominal walls and to the pubes may, by their contraction, move onwards the testicle and peritoneum, because they spring from definite fixed points, but the part which arises from the bottom of the scrotum seems entirely deficient in this respect. Doubtless the scrotal fibres influence the ultimate destination of the gland, but properly prepared specimens show that the actual work of transition is performed by a band of fibres which originates in the perinæum. This is exceedingly well displayed in an infant in whom I found a congenital hernia of the cæcum. In this case the perinæum is occupied by a quantity of unstriped muscular tissue, continuous behind, with the external sphincter and tissues over the tuber ischii, whilst in front its fibres mingle with those of the scrotum, and those which have been mentioned as passing up the posterior wall of the hernial sac to the cæcum. It is not impossible that in this case the dissection was facilitated by the muscle being hypertrophied. It is never easy to follow bands of unstriped muscle with the scalpel, and although the fœtal perinæum always contains them in abundance, it would be rash, without the aid of microscopic sections, to make explicit statements concerning them. However, the combined methods show that this portion of the gubernaculum after emerging from the perinæum is attached to the extremity of the processus vaginalis, the testicle, and

epididymis, and, moreover, that its fibres extend up the posterior surface of the processus vaginalis towards the peritoneum which lines the back of the abdomen. Clearly these are the muscular bands which have been already (p. 502) notified in the plica vascularis.

The preceding statements derive a certain degree of support from the fact that when the testicle exceeds its proper excursion, and passes into the perinæum, it has been seen attached to the tuberosity of the ischium by a band which required division before replacement into the scrotum could be achieved. Both Cloquet[1] and Mr. McCarthy[2] mention a case of this sort, and Mr. Treves has informed me of a similar one under his care. It is not impossible that the perineal fibres of the gubernaculum may in a degree persist throughout life, for in an exceedingly well-developed subject the subcutaneous tissue in that region contained large quantities of unstriped muscle-fibres.

With regard to the part of the gubernaculum testis which extends up the back of the processus vaginalis and into the plica vascularis, it is interesting to note that, as Cruveilhier[3] points out, the spermatic cord contains numerous longitudinal bands of unstriped muscle, which he calls the "internal cremaster." I would identify these as being the upward prolongation of the gubernaculum testis, whose importance in relation to infantile hernia will be shown in what follows. Before discussing this branch of the subject, a last word may be spoken upon the question of the locomotion of the peritoneum.

It may be remembered that it has been repeatedly said that only the serous membrane which clothes the back of the abdomen moves towards the groins. But before this is accepted it is necessary to solve the question why other portions are not involved. The problem seems purely anatomical. The peritoneum which lines the inner surface

[1] Loc. cit., p. 24, 5. This case was verified by dissection.

[2] Loc. cit., p. 1606.

[3] Cruveilhier, 'Traité d'Anatomie,' 1874, vol. ii, p. 381, fig. 253.

of the transversalis fascia and muscle is, both in the fœtus and in the adult,[1] so closely attached to those structures that its displacement is practically impossible. In this situation in the fœtus, sub-peritoneal tissue is almost absent, and the serous membrane is evenly distributed and devoid of pleats and folds. The contrary is the case with that which lines the iliac fossæ and back of the abdomen, for in this situation, as John Hunter[2] points out, its laxity is so great and its connections so loose, that ample folds may easily be seized and dragged in any direction. Histological specimens show that everywhere in the region of the psoas muscle the serous sac is underlaid by a great quantity of the most delicate connective tissue, and that an ample cushion of this is prolonged behind the advancing processus vaginalis into the scrotum. A final reason for the displacement of this particular part of the peritoneum is that the fibres of the gubernaculum are especially distributed to it.

Before endeavouring to apply these anatomical and developmental data to infantile hernia, perhaps the most important may be recapitulated.

A. That the lowest attachments of the gubernaculum are in the perinæum.

B. That the gubernaculum is inserted into, and draws the processus vaginalis into the scrotum.

C. That the gubernaculum is prolonged above the testicle to the peritoneum of the posterior wall of the abdomen, and produces an extensive locomotion of it.

If we now return to inspect the various specimens of infantile hernia which have been mentioned, it is palpable that either a fold of peritoneum, or a well-marked fasciculated band of tissue extends from the upper part of the epididymis to the inferior extremity of the hernial sac.

[1] Cloquet makes a similar observation as regards adults, 'Recherches Anatomiques sur les Hernies de l'Abdomen,' p. 44.

[2] 'Observations on Certain Parts of the Animal Œconomy,' by John Hunter, 1786. A description of the situation of the testicle, p. 8.

This fold is exceedingly well shown in a specimen of infantile hernia which is in the museum of St. Thomas's Hospital (*v.* Fig. 4), and, owing to the manner in which it arises at the upper end of the epididymis, there is not the slightest difficulty in recognising it as the remains of the plica vascularis. Under ordinary circumstances that reduplication of serous membrane almost entirely disappears, but an examination of the various specimens of congenital hernia in the London museums shows that it has a very great tendency not only to persist, but to attain considerable size and stretch far up the posterior wall of the sac. This point is clearly shown in one of Camper's plates[1] and in a specimen of congenital hernia which I obtained from a pig. It is of considerable practical importance because the fold indicates not only the position of the spermatic vessels, but also distinguishes certain adhesions which are found in congenital hernia. The plica vascularis has already been mentioned in connection with cæcocele, and its relation to the disease has been noted. It seems unnecessary to say at length how essential a knowledge of the structure is to the practical surgeon.

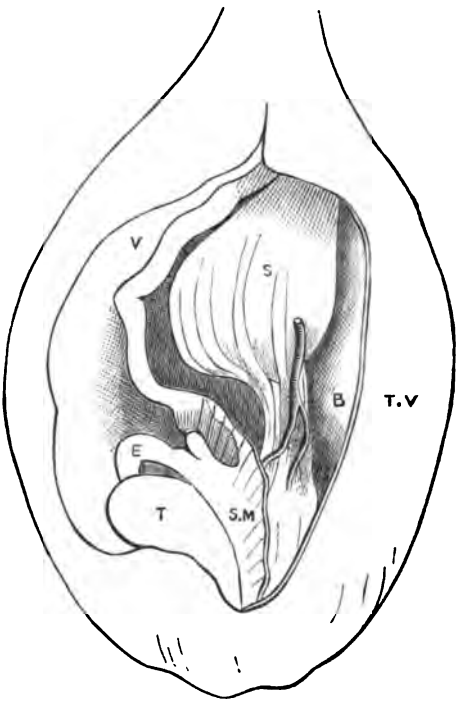
In reading accounts of operations upon congenital herniæ one is struck by the frequency with which adhesions of the gut to the back of the sac, and to the testicle, are mentioned, and often the significant remark is added that when the adhesion was severed, the spermatic vessels were divided.[2] If an opinion may be formed from morbid anatomy specimens this disaster may be avoided by simply ascertaining whether the fold or adhesion is the plica vascularis, and to decide this question it is only necessary to trace the band towards the testicle and observe its relation to the epididymis. After this digression the relation of the plica vascularis to the pathology of infantile

[1] Camper, 'Icones Herniarum,' ed. by S. J. Soemmerring,' 1801, Tab. iii, figs. 3 and 4.

[2] E. g., Pott's 'Chirurgical Works,' vol. ii, p. 159. 1779; also Vidal, 'Traité de pathologie Externe,' tome iv, 1861.

hernia may be resumed. It has been stated that Wrisberg and others consider the fold, which I have ventured to call the plica vascularis, an important factor in the causation of congenital hernia of the cæcum and sigmoid flexure, and

FIG. 8.



Specimen of "encysted hernia" in the Museum of St. Mary's Hospital, Sp. C. D. 20. Shows band passing from epididymis to bottom of sac. The spermatic artery is seen amongst its fibres. The vas deferens passes over sac and was probably at one time closely attached to its walls.

T, testicles; E, epididymis; V, vas deferens; S, hernial sac; B, band with spermatic artery upon it; S. M. Cut edge of serous membrane.[1]

since it is present in this case of infantile hernia (Fig. 4), it might be supposed to have something to do with its

[1] I am indebted to the kindness of Mr. E. Owen and Dr. Silcock for permission to examine and draw this specimen.

formation. Before accepting this inference the absence of the plica vascularis in the case of congenital cæcocele already mentioned (p. 505) entails caution and suggests that the fold, in itself, need not be an essential cause. However, it may be remembered that in its place a quantity of muscular fibres and fibro-areolar tissue passed upon the posterior wall of the hernial sac to the cæcum and probably performed the rôle which, in other cases, has been assigned to the plica vascularis. The pertinence of these remarks will be clearer as the peculiarities of certain cases of infantile hernia are investigated. In the two specimens which are depicted in Figs. 5 and 8, the plica vascularis is not apparent, having, I think, been removed; but, in its place, a strong fasciculated band extends from the epididymis to the lower extremity of the hernial sac. The drawings show that the spermatic artery is intimately associated with this structure, which is proved by the microscope to consist of unstriped muscle-fibres and fibro-areolar tissue. In the St. Mary's specimen the origin of the muscular fasciculi may be traced far down the back of the epididymis, possibly to the scrotum, and in either case they terminated above upon the inner wall of the hernial sac, many of them ascending between the two layers as far as its neck. If the relations of these muscular fibres to the epididymis, spermatic vessels and serous membrane be compared with those which have already been attributed to the upward prolongation of the gubernaculum testis the likeness is manifest, and without doubt they are identical structures. The moment it has been admitted that the band of muscle-fibres, which extends from the epididymis to the sac of these infantile herniæ, is part of the gubernaculum, an explanation of the pathology of that disease is possible. I have already endeavoured to prove that the muscle in question has a most important influence in producing the processus vaginalis and in drawing down the peritoneum, and, if this has been allowed, there can be little difficulty in conceiving that it may, under certain circumstances, produce an additional sac. Before adduc-

ing evidence to support this proposition a circumstance which is common to congenital and infantile hernia may be commented upon. Under ordinary conditions the processus vaginalis, after it has served for the transition of the testicle, ceases to grow and develop except at its lowest part; which, stimulated by the presence of the testis, becomes larger and thicker. However, it occasionally happens that the processus vaginalis, instead of undergoing those retrograde atrophic changes, grows and develops, and its lumen, instead of ceasing to exist, increases. When this happens the enlargement is not confined to the serous membrane alone, but, as specimens in the Dupuytren museum show, its blood-vessels, and in all probability other structures, participate. In this way the persistence of the plica vascularis in congenital hernia, being part of a general effect, may be explained; and it is not unlikely that the hypertrophied condition of the upper part of the gubernaculum (internal cremaster) in cases of infantile hernia is related to it. Long ago Cloquet put it upon record that the gubernaculum could create[1] by its traction the sac of an ordinary hernia and Sir William Lawrence[2] testifies to the importance of this observation by quoting it *in extenso*. If this be so, there is no difficulty in believing that the gubernaculum assists in the production of the sac of an infantile hernia. The morbid anatomy of the disease points strongly to the probability of this assumption. The portion of peritoneum from which the sac is formed, that which lines the back of the abdomen, has already been shown to be loose and easily displaced, and, moreover, it has been affirmed that normally the gubernaculum is inserted into it. Therefore, from an anatomical point of view, the idea is tenable. But before accepting this conclusion, that the sac of infantile herniæ is caused by the traction of the gubernaculum testis, the specimens themselves ought to be examined to see whether they lend any support to it. The following points may be noted: A, that the sac is

[1] Cloquet, 'Causes, &c., des Hernies,' p. 23, *et seq.*

[2] Lawrence on 'Hernia,' p. 19, *et seq.*

always closely related to the posterior wall of the open processus vaginalis and usually bulges into it; B, that the sac is formed from the loose and yielding peritoneum of the back of the abdomen; C, that a band of muscular fibres closely connected with the spermatic vessels is inserted into the inferior extremity and surface of their sac wall. Although these are cogent reasons yet it might be anticipated that a sac, which owes its birth to tractive force, would betray its origin by its conical shape. None of the infantile herniæ which have been mentioned are particularly pointed. In one case (Guy's 2497, Fig. 5, p. 495) the posterior wall of the hernial sac exhibits a suggestive pouch which descends behind, and parallel to, the main sac, but in other respects their shape is very like that ordinarily produced by pressure from within. These facts do not forbid the supposition that at the commencement these sacs may not have been originated by the gubernaculum and afterwards modified by pressure, and a specimen which I have dissected countenances this view (Specimen 2140^{3}, St. Bartholomew's Hospital Museum). In it the processus vaginalis was represented by a long tube which extends from the internal abdominal ring to just above the epididymis. This tube communicates with the general cavity of the peritoneum by a small aperture, a quarter of an inch in diameter, which occupies the usual position of the internal abdominal ring external to the epigastric artery. A probe introduced into this opening showed that the processus vaginalis was occluded an inch from its upper end, but in the remainder of its extent its cavity was almost half an inch in diameter, above, and one and a half below. Behind the superior part of this serous tube a hernial sac protruded from the peritoneum in such a way that its anterior wall bulged slightly into the cavity of the processus vaginalis. Attached to the lower extremity of this protrusion and to its posterior wall were strong bands of unstriped muscle-fibre intimately related to the spermatic vessels. The end of the sac to which these were attached was conical and sharply pointed.

It seems hardly requisite to enumerate the reasons why this case should be included in the category of infantile hernia, and it clearly shows by its shape that the hernial sac was caused by the traction of the gubernaculum testis. This specimen also demonstrates that in infantile hernia the processus vaginalis need not necessarily communicate with the cavity of the tunica vaginalis, because in it the latter was shut off from the former, in the same way as in funicular hernia. This fact seems also to be displayed by other specimens of infantile hernia in the museum of St. Bartholomew's Hospital (Sp. 2140*c* and 2140*a*), but as I have not yet dissected these no other assertions will be made concerning them.

The conclusion arrived at, after studying these data, is that the sac of an infantile hernia owes its origin to the action of the gubernaculum testis, but that afterwards it may be considerably modified by pressure from within. In this way may be explained a circumstance which seems to militate against many of the previous assertions. A glance at Fig. 6, p. 498 shows that the sac of this hernia protrudes in the open processus vaginalis like a cyst pendent from the ring and that there is no trace of muscular fibres reaching from its extremity to the epididymis. However, when this beautiful specimen is viewed in profile it is clear that its attachment to the posterior wall of the vaginal process is quite an inch long, and although no muscle-fibres are attached to the bottom of the sac, numerous bands may be perceived running upwards behind the serous membrane (vaginal process), and when they arrive at the fornix, which is formed by the junction of the vaginal process with the outer layer of the hernial sac, they turn forwards and insinuate themselves between the two layers of serous membrane which constitute its walls. The construction of the sac of this hernia, and the condition of the peritoneum at its neck have already been discussed, *v. a.*, and it has been decided that it belongs to the infantile variety. This being the case, although it is evident that pressure from within has profoundly affected its

sac, yet it cannot be denied but that it may have had something to do with its beginning.

Before concluding these observations reference may be made to a point which has not yet been touched upon. In nearly all the cases of infantile hernia which have been mentioned the upper edge of the hernial sac is formed by the posterior margin of the aperture by which the processus vaginalis communicates with the cavity of the peritoneum. It seems natural to ask by what means this acquires its immobility. In the St. Mary's specimen this portion of serous membrane is exceedingly thick and strong, and attached by a species of alæ to the peritoneum of the front wall of the abdomen, which has been shown to be comparatively immobile. In the specimen in the museum of St. Bartholomew's Hospital which has just been described a not dissimilar condition exists, and besides the serous membrane exhibits many old scars and thickenings; but, at the present, it would be premature to express any definite opinions upon this point, for there is reason to think that the neck of the sac may sometimes be produced in a different manner. In conclusion, perhaps, I may be permitted to recapitulate the results arrived at by this inquiry:

a. That the London museums contain no specimen of encysted hernia such as is usually described.

b. That the various specimens designated by that name belong to the infantile variety.

c. That the latter owe their origin to the tractive power of the gubernaculum testis.

(For a report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' New Series, vol. ii, p. 118.)

ON A CASE OF MULTIPLE NEUROMATA.

BY

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Received February 9th—Read June 8th, 1886.

MARGARET E— æt. 30, admitted into the General Hospital, Birmingham, July 18th, 1885.

History.—Four years ago was treated as an out-patient for what was then considered to be an enlargement of the cervical glands on the right side. An abatement in the size of the tumour apparently resulted, and no further increase in size took place until three months before admission. At first some large glands also existed on the left side of the neck, but these gradually disappeared. The patient now sought advice at the hospital because the tumour was growing, and caused pain down the arm on the affected side.

On admission.—There is a tumour as large as a duck's egg in the right posterior triangle of the neck, movable and seeming to all intents and purposes of a lymphomatous nature. The patient was short and stout, and slightly anæmic, but the general health appeared normal, and the various functions of the body were naturally performed.

Operation, July 24th.—A longitudinal incision was made over the growth, and, on reflecting the skin and fascia, a large nervous cord was found running over its upper

surface and required to be dissected off. The tumour itself extended deeply, dipping down behind the clavicle, and at its upper part was found attached by a pedicle, the thickness of the little finger, to the vertebral column.

During manipulation this pedicle was torn across close to the spine, and the step was followed by a gush of blood. A rounded aperture into which the tip of the finger could be inserted was left by the removal of the pedicle, and had to be plugged by a strip of boracic lint to stop the hæmorrhage. The wound was then drained and its edges approximated.

The evening temperature was 101° F., and the patient complained of violent headache.

July 25th.—Patient was semi-conscious, but could be roused, when she complained of her head aching. The arms and legs were constantly tossed about. There were twitchings of the facial muscles, and the urine passed involuntarily. Pupils slightly contracted and sluggish; the temperature varied between 102° and 103·4° F.

Next day the patient was quite sensible and remembered nothing of the previous day. On dressing the wound and removing the plug about two drachms of a clear-looking fluid escaped.

July 28th.—There was a rigor, followed by delirium and marked rigidity of the neck and head. This condition continued until July 31st, when muscular tremors and slight clonic spasms became marked. The pupils were widely dilated, and coma supervened, and the patient died in the evening.

Post-mortem August 1st.—Body very bloodless; rigor mortis slight.

Neck.—An incision three inches long existed parallel to the edge of the sterno-mastoid muscle, in the right posterior cervical triangle. This opened into an irregular cavity, one and a half inches in diameter, that led backwards and inwards to the spinal column, and at its very bottom was a round intervertebral foramen (fifth) empty of its nerve and containing pus. All the structures bound-

ing this cavity were thickened and adherent from inflammatory exudation.

Nervous system.—Brain weighed 52 oz. The membranes were smooth and shining; no sign of meningitis either on the vertex or at the base. The ventricles were distended with a thin clear fluid, and their walls were softened. This was the only abnormal change found in the brain-substance.

The *spinal cord* and plexuses were removed entire. The posterior surface of the cord was deeply congested, and thinly coated with a soft layer of dirty, yellowish-brown fibrin. This began at the fifth cervical nerve, and extended down the cord, but it did not pass to the front or ascend to the brain. There was a considerable quantity (half an ounce approximately) of thin semi-purulent fluid in the cavity of the arachnoid, which escaped when the latter was opened. The pia mater on the front of the cord was deeply congested, but there was an entire absence of lymph. The cervical enlargement was soft and pulpy, especially opposite the sixth nerve, and, on section, the substance was discoloured, the white matter being of a greyish tint, and the grey matter less defined than usual. The fifth cervical nerve was discoloured and thickened on the right side from inflammatory changes. The sixth nerve had been torn off, the root giving way inside the dura mater, so that the ganglion went with the torn portion. The ends of the anterior and posterior roots were found within the dura mater. All the nerves that could be examined were found to be irregularly enlarged. Surrounding them were various sized tumours contained within the nerve-sheath, and apparently having the nerve running through them like an axis. Most of them were fusiform, a few globular, and, on section, they appeared white, glistening, semi-translucent, and extremely firm. The nodulation began as soon as the nerve left the dura mater, and was first seen in the ganglion of the root which, all down the cord, was greatly enlarged.

Inside the dura mater the nerves were quite normal.

The trunks of the nerves were much increased in size by a sort of diffusion of the tumour, so that, for example, the sciatic was one and a quarter inches broad and proportionally thick, and the anterior crural half as large again as the normal sciatic. Even the small nerves, *e. g.* the genito-crural, were affected, and on them, the nodules were much larger in proportion to the diameter of the nerve, than was the case with the larger ones. The sympathetic nerves were similarly affected, and the fine filaments in the rectum could be easily traced by means of the nodules. They could also be seen beneath the mucous membrane of the tongue and the pharynx.

The pneumogastrics were equal to a penholder in size. The phrenic nerves appeared like a string of dahlia roots. The various thoracic and abdominal viscera were healthy.

Similar cases of so-called multiple neuromata appear to be somewhat rare. Lebert has collected seventeen cases, and Prudden,[1] of New York, has extended these to forty-one. From such records, the clinical histories being extremely meagre in seven, the following facts may be deduced:

I. The male appears to be more prone to this development than the female. The sex is recorded in thirty-two instances, and of these twenty-four were men, eight were women.

II. The middle period of life is most liable to the affection, but in some of the cases Prudden's opinion is that the tumours were undoubtedly congenital.

III. The duration of the disease has not been determined. In twenty-three cases, where the age at death is stated, the fatal termination occurred, on an average, between thirty-three and thirty-four years.

What Lebert terms the second stage of development, and this appears to be the period of pronounced swellings, is stated to be, five or six months.

[1] American Journal of Med. Sc.,' July, 1880.

IV. Clinically no constant symptoms are manifest in cases of multiple neuromata.

In twenty-six instances where the history is fully enough reported, twelve had no symptom pointing to a nerve lesion.

In three there was more or less paralysis, but this by no means in proportion to the size and number of the tumours.

Pain was only experienced in thirteen patients; this varied much; in some it is described as being spontaneous, in others it was elicited by pressure or atmospheric changes. This absence of pain seems remarkable, considering that both the mixed and sensory nerves were covered with tumours. Typhoid fever appears to be badly borne in this class of case, five deaths being attributed to it in a mortality of twenty-seven.

Three patients died of phthisis; in one recorded by Dr. Wilks,[1] the writer thinks that it is possible that the condition was due to the lesion of the pneumogastric inducing the pulmonary changes.

In many cases, nutrition of the body is reported to have been interfered with, yet on post-mortem examination no organic disease of the viscera was found. Most authors agree that the prognosis in this disease is unfavorable.

V. The tendency of the condition is to appear in several members of a family. Nicaise[2] thinks there is sufficient evidence to show that it is frequently congenital and hereditary. Hitchcock[3] has reported cases in which the mother, her son and daughter all exhibited multiple neuromata. Generisch[4] cites an instance in which the patient, whose mother had suffered from numerous tumours diagnosed as neuromata, died of pneumonia. At the post-mortem, tumours of various sizes were found on nearly all the nerves of the body. Four years

[1] 'Transactions of the Pathological Society of London,' vol. x.

[2] 'International Encyclopædia of Surgery,' vol. iii.

[3] 'American Journal of Med. Sciences,' vol. xliii, 1862.

[4] Virchow's 'Archiv,' Band 49, 1870.

afterwards the brother of the preceding case died of tetanus, and neuromata were then found to exist everywhere. Both vagi and the phrenics were affected. The roots of the spinal nerves were normal.

Bruns[1] reports a case in which death was caused by hæmorrhage from the carotid artery. Many tumours, some the size of a pigeon's egg, were found on the nerves. The patient's brother had congenital elephantiasis with plexiform neuromata about the head and neck, and the mother is said to have had wart-like tumours in the skin.

VI. Operative interference is badly borne. This is exemplified by the following cases:

(A) One of the tumours removed from the left radial nerve. Death in five weeks from pyæmia.

(B) Amputation of right leg for ulceration and gangrene of toes. Died in two days of pneumonia.

(C) Tumour near clavicle the size of a hen's egg and another small one near the lip were enucleated and did not return. According to the statement of the patient the tumours in the other parts of the body increased in number more rapidly after the operation.

(D) Removal of tumour, six and a half by three and a half inches in size, from the right ulnar nerve, the nerve itself being severed in the operation. The wound healed; a year later disarticulation at the shoulder-joint was performed for a return of the growth. The stump did not heal. Death from exhaustion seven months later.

(E) Removal of tumour the size of a clenched fist. Vagus divided. Died on the tenth day, hæmorrhage taking place from ulceration of the carotid.

(F) Attempt made to remove a tumour from the back. Died of pyæmia.

(G) A portion of the lesser sciatic nerve excised in an endeavour to check the growth of many tumours corresponding to the branches of the nerve. Wound healed by suppuration. After four months many of the swellings disappeared and the rest gave no trouble.

[1] Virchow's 'Archiv,' Band 50, 1870.

VII. The tendency of the disease to become malignant.

This appears to be rare. In Hitchcock's third case the tumour removed from the ulnar nerve, after existing for upwards of twenty years, presented on section the characters of a doubtful neoplasm. A year later, after amputation of the limb for its recurrence, it was certified to be of an encephaloid nature, and the patient died a few months later with a return in the cicatrix.

In Genersich's[1] case, multiple tumours having existed for some time, ten weeks before death a rapidly growing neoplasm appeared in the right buttock. Examination after death showed that some of the tumours were fibromata, some sarcomata, and others myxomata.

In both these cases there was a hereditary tendency to neuromata, and the mother of the second case had carcinoma of the mamma.

VIII. The position of the tumours.

In twenty-seven cases the peripheral nerves were affected.

In ten cases special groups of nerves were implicated.

In sixteen cases the sympathetic and in twenty-two the vagi were involved.

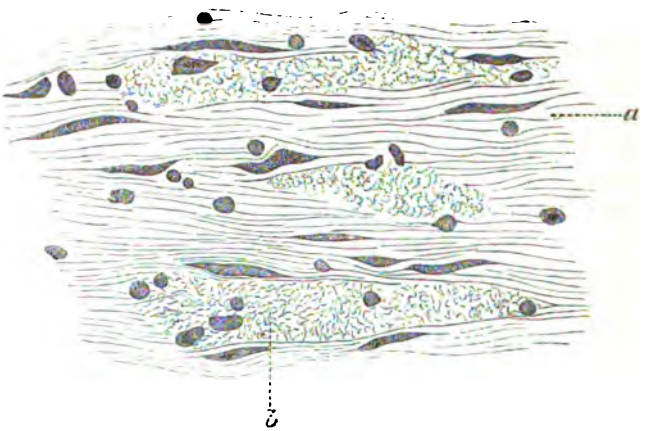
As a rule, however, special nerves appear to be unaffected and the nerves of the hands and the feet are free from the lesion.

Microscopic Examination of the Tumour itself.

The tumour is composed of anastomosing and branching bundles of white fibrous tissue, which intersect one another at varying planes. Between the fasciculi are embedded numerous fusiform, oval, and round cells, resembling the embryonic connective-tissue corpuscles. No elastic fibres are to be seen and no well-defined stellate or branching connective-tissue cells. There is no evidence of fully developed nerve-fibrils in any of the sections. (See Woodcuts on page 524.)

[1] Virchow's 'Archiv, Bd. 49, 1870.

a. Spindle-cells and connective tissue cut transversely, with some round-cells; *b*, spindle-cells in delicate connective tissue.



a. Spindle-cells and fibrous tissue cut longitudinally, with a few round-cells; *b*, spindle-cells and connective tissue cut transversely, with some round-cells.

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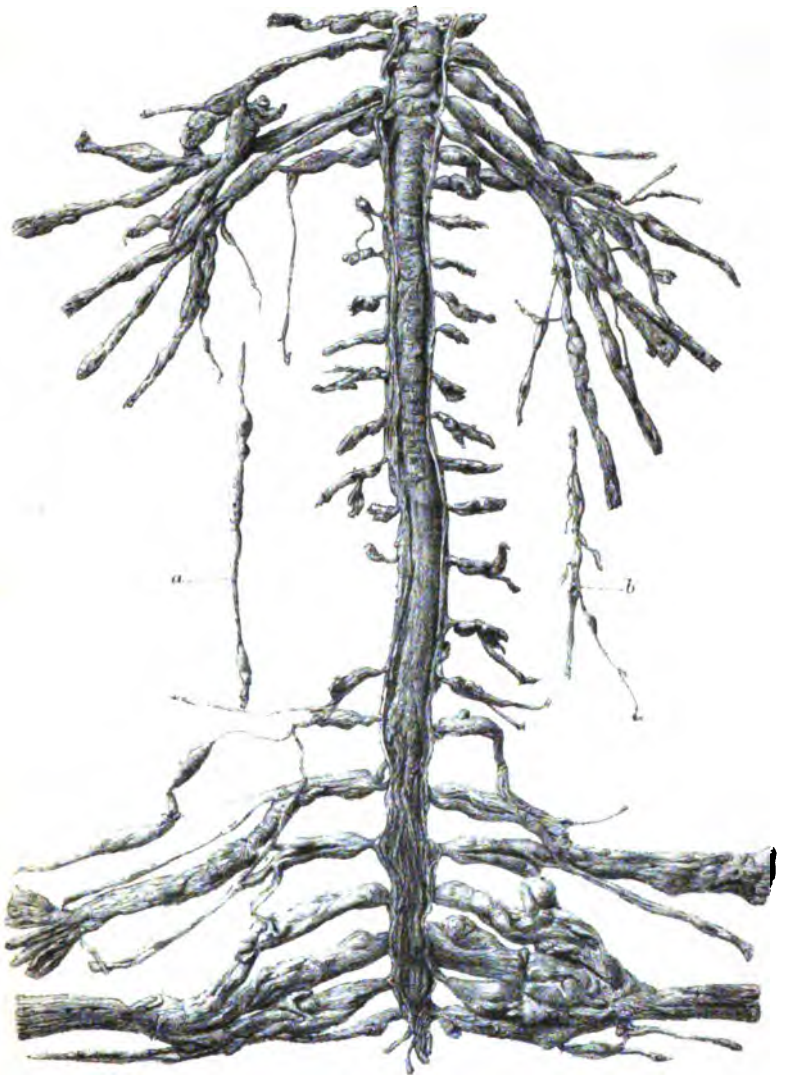
(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' New Series, vol. ii, p. 125.)

DESCRIPTION OF PLATE XIV.

Multiple Neuromata. By T. F. Chavasse, F.R.C.S.

a. Smallest splanchnic.

b. Genito-crural.



Mintern Bros. lith.

SOME STATISTICS OF PNEUMONIA,

WITH ESPECIAL REFERENCE TO THE

RELATIONS OF DELIRIUM AND TEMPERATURE.

BY

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Received March 9th—Read June 8th, 1886.

THE following statistical tables have been drawn up from an investigation of the cases of pneumonia recorded in the University College Hospital case books during the past twelve years. I am indebted to Sir William Jenner, Dr. Russell Reynolds, Dr. Wilson Fox, Dr. Sydney Ringer, Dr. Charlton Bastian, and Dr. F. T. Roberts for permission to make use of cases that had been under their care.

The plan that I have pursued has been to make a concise abstract of the cases, paying every attention to the notes on the temperature and state of the nervous system. From these abstracts a table, not here presented, was constructed, and its various factors have been carried through a kind of permutation and combination, the results of which processes are here recorded. I have in a few places ventured on some suggestions, and notably in connection with the unexplained circumstance that delirium is so frequent with pneumonia of the upper lobes of the lungs.

TABLE I *gives age and sex of all the cases.*

Age. Years.	Male.	Female.	Total.	Percentage.
1—10	41	14	55	27·5
11—20	24	17	41	20·5
21—30	30	16	46	23·0
31—40	21	6	27	13·5
41—50	16	4	20	10·0
51—60	6	2	8	4·0
61—70	1	1	2	1·0
	139	60	199	

TABLE II, *showing site of lung affected and the sex of all the cases.*

	Right lung.		Left lung.		Both lungs.
	Upper lobe.	Lower lobe.	Upper lobe.	Upper lobe.	
Male	9	59	4	47	20
Female	2	26	4	15	13

The right lung alone was the seat of pneumonia in 96 cases, or a percentage of about 48. Bleuler gives the percentage at 52. The left lung alone was affected 70 times, or 35 per cent. Bleuler gives 32 per cent. There was double pneumonia 33 times, or 16 per cent., which also agrees with Bleuler.

TABLE III, *showing the number of cases according to age, sex, and site of pneumonia.*

	Right lung.		Left lung.		Both lungs.	
	Male.	Female.	Male.	Female.	Male.	Female.
1—10	16	5	21	5	3	4
11—20	12	6	10	5	4	4
21—30	17	7	6	5	5	3
31—40	9	1	8	2	5	2
41—50	10	3	4	1	2	—
51—60	5	1	1	1	—	—
61—70	—	1	1	—	—	—
	69	24	51	19	19	13
	93		70		32	

Fallacies of statistics.—No one can be more aware of the fallacies of statistics than I am. To reject statistics altogether, though perhaps the most logical proceeding, appears to me to be unadvisable. That statistics have introduced many false facts into medicine I do not doubt, but I cannot but believe that we have also benefited by them.

The statistics which are here presented seem to me to be suggestive rather than positively instructive, and will serve the purpose more of indicating lines of future study than of laying down fresh propositions. At the same time, as a solid contribution to our collection of facts concerning pneumonia, the author submits that this paper must necessarily possess some value in and of itself.

The apparent discrepancies in numbers is to be explained by bearing in mind that all the cases were not always available for every table.

The cases are all cases of *lobar* pneumonia. The majority of the cases are *simple* ones of *primary* pneumonia. A few cases are interspersed in which there were marked complications, or in which acute pneumonia supervened on another disease. I have retained these cases for comparison and with a view to their throwing light on the symptoms in primary pneumonia.

From the total number of cases, 199, we subtract the following (17) in which the pneumonia was not the only disease:

1. Case 54, man, aged 23, pneumonia of the whole of the right lung complicated by pleurisy and acute Bright's disease, fatal.

2. Case 55, woman, aged 37, pneumonia of left lower lobe, mitral disease, recovery.

3. Case 84, man, aged 20, pneumonia of right lung, complicated by peritonitis, death.

4. Case 121, delirium tremens, man, aged 27, Bright's disease, fatal.

5. Case 128, man, aged 36, right base affected, Bright's disease, recovery.

6. Case 129, female, aged 16, double pneumonia, rheumatic fever, recovery.

7. Case 158, female, aged 15, rheumatism, double pneumonia, fatal.

8. Case 189, male, aged 62, complicated by pericarditis, pleuro-pneumonia of left lower lobe, fatal.

In these 8 cases delirium was present.

9. Case 2, female, aged 42, left lower lobe pneumonic, mitral disease, death.

10. Case 4, female, aged 19, double pneumonia, Bright's disease, death.

11. Case 32, infant, lethargic, aged 1, pericarditis, left lung pneumonic, fatal.

12. Case 35, male, aged 11, lethargic, left lower lobe pneumonic, rheumatic fever, recovery.

13. Case 64, male, aged 6, right lower lobe pneumonic, meningitis, mental dulness, death.

14. Case 75, male, aged 50, left lower lobe pneumonic, pericarditis, fatal.

15. Case 101, female, aged 29, mitral disease, double pleuro-pneumonia, recovery.

16. Case 102, female, aged 29, rheumatic fever, mitral disease, right lower lobe pneumonic, recovery.

17. Case 188, male, aged 55, right lower lobe pneumonic, pericarditis, death.

The above 9 cases had no delirium.

Of 182 cases of primary pneumonia there were 56 in which delirium was present, or a percentage of 30.

Heinze ('Archiv der Heilkunde,' 1868, p. 49) has studied the relations of marked mental symptoms to the temperature in pneumonia and has arrived at the conclusion that the mere pyrexia had little or nothing to say in the matter. My investigations tend in the same direction. But the consideration cannot be lost sight of that prolonged pyrexia and high transitory fever must

exercise some direct and indirect deteriorating influence on the grey matter of the brain and spinal cord, and must therefore predispose to delirium and other signs of exhaustion of the nervous matter.[1]

Of 17 available cases sometimes (4) the delirium coincided with the greatest rise in temperature; rarely (2) it preceded the acme of fever, and most frequently (11) the delirium came on with the fall of temperature.

Heinze draws attention to the much greater frequency of delirium, or rather, marked mental change in pneumonia of the upper lobe of the lung. Of 317 cases the upper lobes were involved 117 times; the lower lobes were alone affected 200 times. Of the 98 cases showing delirium, 47 were cases of pneumonia of the upper lobe and 51 of the lower lobe. The contrast is made more striking by a detailed statement of the facts (loc. cit., p. 57).

I think it safest and least liable to error if a comparison be made between cases which affect the upper lobe alone and those which affect the lower lobe alone.

Thus, of 25 available cases in which the upper lobes were alone diseased, I find that 12 are reported as delirious, or a percentage of 48, which is 7 per cent. higher than Heinze's estimate. When the lower lobe was alone involved in 110 available cases, I find that there was delirium 28 times, or a percentage of 25·5. These results are practically identical with those of Heinze.

Liebermeister believes that one of the reasons for the above difference is to be found in the longer duration of pneumonia of the upper lobe. Heinze adduces evidence to show that this conclusion does not hold good. My notes so far as they go support the contention of Heinze.

Thus, in Case 8, the onset was on November 6th, and the fever had disappeared by the 13th; the temperature was frequently 105°; the unconsciousness lasted till the

[1] See a paper by author on "Reflex Actions, &c.," 'The Lancet,' vol. ii 1885.

9th; there was some "after" fever on the night of the 13th, which had ceased by the 15th; the temperature remained quite normal after the 23rd. Case 9 began on March 12th and ended on the 19th; no mention was made of delirium.

Case 58, the man had suffered from epilepsy; it was a fatal case of pneumonia of the right apex which began on July 30th and ended on August 9th. Case 69 lasted only seven days (August 1st to 8th). Case 78 began on May 9th and terminated on May 17th. Case 129 began on May 22nd and ended on May 29th. Case 130 lasted from August 22nd till August 30th. Case 140 commenced on February 9th and ended fatally on February 13th.

In several other instances there are no notes to fix the date of onset, but the course and height of the fever on admission and attendant circumstances would lead one to suppose that the duration was not abnormally long. Further, the temperature of cases of pneumonia of the upper lobe alone does not appear to be higher than in pneumonia of the lower lobe. And though my notes show that the temperature was generally high and sustained in cases of pneumonia of the upper lobe, yet a comparison of the number of cases according to site of disease and temperature gives no certain indication that there is any remarkable difference in the degree of pyrexia in pneumonia of the upper as contrasted with that of the lower lobe.

Heinze brings forward some figures to show that the rate of mortality in pneumonia of the upper lobe is higher than in pneumonia of the lower lobe. Taking again only those cases in which the disease was confined to the upper or the lower lobe my statistics give the following results: — Five deaths in 25 cases of pneumonia of the upper lobe alone, and ten deaths in 110 cases of pneumonia of the lower lobe alone, or a percentage of about 20 in the former and 10 in the latter. The numbers are small, but the difference is great. Of the 12 cases of delirium with pneumonia of the upper lobe, but 2

proved fatal (16·6 per cent.); of the 28 cases of delirium with pneumonia of the lower lobe, 4 proved fatal (14·3 per cent.). The difference here is not nearly so great as that given by Heinze, whose numbers are 34 per cent. and 21·5 per cent. respectively. This author seeks for a satisfactory explanation of the greater frequency with which delirium occurs in pneumonia of the upper lobes and finds none. He examines the age, sex, drinking habits, month of the year of all the cases of delirium, and all to no purpose.

After a careful survey of the statistics that I have collected, I have arrived at the following position:

The determination of delirium in any particular case probably depends on at least several factors or elements in the case. The age of the patient probably has some influence, but I think not much, except in this way. Delirium is disorder of the intellectual faculties and inextricably mixed up with the functions which are engaged in the process of speech. Infants, therefore, are incapable of delirium in the ordinary sense of the term, for the reason that they are not in possession of the organised elements on which intellectual actions depend. But that the mental or cerebral functions are greatly disturbed in infants a glance at the collected facts readily proves. A little consideration will show also that sex can have but little to say in the matter.

Previous habits and social conditions probably play some share in the production of delirium. Some of the most powerful causes are alcohol, tea, and tobacco. The prolonged and excessive use of these articles of consumption probably deteriorates considerably the structures on which intellectual processes depend.

Unquestionably a neuropathic disposition, however brought about, would be a potent element in the causation of delirium.

Is there anything special in the nature of pneumonia which tends to produce delirium? I do not think so. I do not think that pneumonia is associated in any way

with the production of any substance which has "deliriant" properties like belladonna.

That some cases of delirium in pneumonia may be dependent on the absorption into the circulation of an autogenetic alkaloid is possible. But the action of alkaloids formed in the tissues in pneumonia (such as have been found by MM. Villiers, Lepine, and Guérin) is unknown.

But one more suggestion I have to make in connection with the greater frequency of delirium in pneumonia of the upper lobes. I make the suggestion that the proximity of the intense inflammation to important and extensive nervous structures in the neck is an element in the explanation. An intense process like lobar pneumonia must influence by radiation the structures in its vicinity. There are the brachial plexus and the cervical sympathetic nerves. The cervical sympathetic watches over the calibre of the arteries supplying the head. I suppose that the arteries supplying the brain are under the dominion of its influence.

Let it be imagined that pneumonia of the apex is capable by its action on the cervical sympathetic of interfering with the supply of blood to the brain. An impairment in the cerebral blood supply, whether as hyperæmia or in the direction of anæmia, must damage or tend to damage the nervous tissues on which cerebral functions are dependent.

Phthisis is well known to be associated with a hopeful state of mind. Phthisis is most frequent at the apices of the lungs. All cases of phthisis are not in a state of hope. Does the difference depend on the site of the lesion? I merely make suggestions, and am fully aware that I am on unsafe ground. Abdominal disease is, as a rule, associated with mental depression. I hardly like to write the following crude attempt at an explanation, as it is open to so many logical objections. Disease of the apices of the lungs irritates the sympathetic and causes hyperæmia of the brain; joyfulness and hopefulness are

said to be associated with increased supply of blood to the brain. Abdominal disease irritates the abdominal sympathetic, opens the floodgates of the abdominal vessels, and drains blood away from all parts of the body, including the brain. A deficient supply of blood to the brain is said to go with mental depression and apathy.

It would be very interesting and might be very instructive if we had some accurate information concerning the relations of delirium to the collective amount of sleep which the patient enjoyed.

TABLE IV *shows the number of cases of delirium at different temperatures (the highest recorded temperature in each case).*

Temp.	98°+	99°+	100°+	101°+	102°+	103°+	104°+	105°+
No. of cases ...	—	—	2	2	6	11	26	9

TABLE V *shows the number of cases without delirium at different temperatures.*

Temp.	98°+	99°+	100°+	101°+	102°+	103°+	104°+	105°+	106°+
No. of cases...	8	1	7	12	19	27	34	14	2

A comparison of these tables appears to show that the number of cases with delirium is largest at the temperature of 104° and 105°. This comparison is rendered more apparent by Table VI, which contrasts nearly equal numbers of cases with and without delirium.

TABLE VI.

Temp.	98°+	99°+	100°+	101°+	102°+	103°+	104°+	105°+	106°+
No. of cases with delirium	—	—	2	2	6	11	26	9	—
No. of cases without delirium...	1·5	1·5	3·5	6	9·5	13·5	17·0	7	1

It would seem, therefore, that the temperature does exercise some, though probably small, influence.

Of the 199 cases there were 42 deaths=about 20 per cent.

TABLE VII.—*Fatal cases with delirium.*

There was	1	„	„	100°+
„	1	„	„	101°+
„ were	4	„ cases	„	102°+
„	5	„ „	„	103°+
„	7	„ „	„	104°+
„ was	1	„ case	„	105°+

TABLE VIII.—*Fatal cases without delirium.*

There were	2	fatal cases when the highest recorded temperature was		100°+
„ was	1	„ case	„	101°+
„ were	3	„ cases	„	102°+
„	2	„ „	„	103°+
„	7	„ „	„	104°+
„	4	„ „	„	105°+
„	3[1]	„ „	„	106°+

From an examination of these tables it seems clear that the presence or absence of delirium exerts no influence on the mortality. A temperature above 105°, whilst not necessarily causing delirium, seems to be of grave significance; the three fatal cases at this temperature were infants, who are incapable of delirium in the ordinary sense of the term.

TABLE IX.—*Number of cases of delirium with recovery at different temperatures.*

Temperature	100°+	101°+	102°+	103°+	104°+	105°+
No. of cases	1	1	3	10	21	9

TABLE X.—*Number of cases without delirium with recovery at different temperatures.*

Temperature...	−100°+	100°+	101°+	102°+	103°+	104°+	105°+
No. of cases ...	4	7	10	18	28	29	14

[1] Case 32, male infant, aged 1 year, highest temperature 106·2°; left lung pneumonic; the child was lethargic.

Case 140, male infant, 9 months, highest temperature 106·8°; right apex pneumonic.

Case 132, female infant, 10 months, temperature 107·2°; double pneumonia.

TABLE XI, *showing the age, highest temperature, and number of Cases of delirium in pneumonia.*

	Age.											
Temp.	1—10		11—20		21—30		31—40		41—50		51—60		Above.
100°+	—	...	—	...	1	...	—	...	—	...	—	...	1
101°+	—	...	—	...	1	...	—	...	1	...	—	...	—
102°+	—	...	2	...	2	...	1	...	1	...	—	...	—
103°+	—	...	4	...	2	...	6	...	1	...	1	...	—
104°+	2	...	7	...	9	...	6	...	1	...	2	...	—
105°+	1	...	—	...	3	...	2	...	—	...	2	...	—
106°+	1	...	1	...	—	...	—	...	—	...	—	...	—

From this table we may state that the third decade, when the temperature goes beyond 104°, seems to be most fertile in the production of delirium. Such a statement is open to several fallacies, and probably means but very little: for an examination of other tables and statistics shows that this period of life and this degree of fever probably yield the greatest number of cases of pneumonia.

The right lung was affected 69 times without delirium, and of these cases 11 proved fatal. The left lung was involved 50 times without delirium, and 7 proved fatal. Both lungs were affected 17 times without delirium, and of these 5 ended in death.

There were 27 cases in which the right lung was affected and the patients were delirious, a fatal termination taking place 9 times. The left lung was affected in 20 cases, delirium being present, and 2 of these died.

Sixteen times the pneumonia was double and the patients delirious; 8 of these succumbed.

The fatal cases of double pneumonia associated with delirium consisted of 2 males in the second decade and 1 female; 1 male and 1 female in the third decade, 1 female in the fourth, and 1 male in the fifth.

Four of these cases were uncomplicated double pneumonia, 1 was complicated with rheumatism, another with slight empyema, another with Bright's disease and delirium tremens. In one of the uncomplicated cases the delirium

was of violent character, with a falling temperature, and in another case of violent delirium the highest temperature recorded was but 102·6°.

Of the 2 fatal cases of pneumonia of the left lung associated with delirium 1 occurred in the fourth and the other in the seventh decade. Of the 9 fatal cases of pneumonia of the right lung associated with delirium 2 occurred in the second decade of life, 3 in the third, 1 in the fourth, 1 in the fifth, and 2 in the sixth.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' New Series, vol. ii, p. 127.)

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